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BRITISH SURGICAL PRACTICE

Under the General Editorship of

SIR ERNEST ROCK CARLING, F.R.C.S., F.R.C.P.
CONSULTING SURGEON, WESTMINSTER HOSPITAL

and

SIR JAMES PATERSON ROSS, K.C.V.O., M.S., F.R.C.S.
SURGEON AND DIRECTOR OF SURGICAL
CLINICAL UNIT, ST. BARTHOLOMEW'S HOSPITAL;
PROFESSOR OF SURGERY, UNIVERSITY OF LONDON

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SIR ERNEST ROCK CARLING
F.R.C.S., F.R.C.P.
CONSULTING SURGEON, WESTMINSTER HOSPITAL
AND

SIR JAMES PATERSON ROSS
K.C.V.O., M.S., F.R.C.S.
SURGEON AND DIRECTOR OF SURGICAL CLINICAL UNIT,
ST. BARTHOLOMEW'S HOSPITAL; PROFESSOR OF SURGERY,
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S. P. MEADOWS, M.D., B.Sc.,
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Senior Surgeon, Westminster
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PERITONITIS**

R. MILNES WALKER,
M.S., F.R.C.S.,
Professor of Surgery, University
of Bristol; Surgeon, Bristol
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MALINGERING

BY S. P. MEADOWS, M.D., B.Sc., F.R.C.P.

PHYSICIAN, WESTMINSTER HOSPITAL; PHYSICIAN TO OUT-PATIENTS, NATIONAL HOSPITAL, QUEEN SQUARE; PHYSICIAN, MOORFIELDS, WESTMINSTER AND CENTRAL EYE HOSPITAL, LONDON

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1. DEFINITION: MALINGERING AND HYSTERIA

28]. Malingering is the deliberate or conscious feigning of illness, whether physical or mental, for personal gain. Pure malingering is probably rare, but exaggeration of the symptoms of an illness is a common clinical experience. A spurious symptomatology may be built up on a relatively minor illness, injury or structural defect.

Malingering is to be distinguished from hysteria, in which the patient also presents symptoms for some personal motive or advantage, but in this case without being fully aware of the mechanism involved. The malingeringer deliberately attempts to deceive others; the hysteric deceives himself, and is assumed to have no clear recognition of the process.

The term hysteria is frequently misused, being commonly confused with the term neurosis, or used synonymously with psychogenic disorder of any type. It is a variety of neurosis and, like its near relation, malingering, can give rise to considerable difficulty in clinical diagnosis, for organic disease can be closely simulated in both disorders.

It is often difficult to distinguish malingering from hysteria. It is indeed likely that the two conditions merge into each other, and that all gradations occur between the deliberate deceit of the malingeringer and the unconscious motivation of the hysteric.

2. MOTIVE

The conscious adoption and subsequent exploitation of symptoms of illness naturally imply a motive. The desire for gain, monetary or otherwise,

overrides truthfulness. In war-time the avoidance of military service and the removal of the subject from dangerous or disagreeable duties, are obvious motives. In civilian life the desire for compensation following industrial or road accidents, resentment against some person or authority, or merely the desire to avoid difficulty or to gain sympathy or attention, may be a precipitating factor both in malingering and in hysteria.

3. PERSONALITY

(1) *In malingering*

Whilst one may argue that malingering might occur in a normal person, provided that the motive was of sufficient importance, it seems likely that in many, if not all, cases of this disorder there is an underlying personality disorder. Apart from his symptoms, the previous history suggests that the malingerer is socially inadequate and emotionally immature. Frequent changes of job, poor consideration for others (both relatives and employers), and little evidence of shame or guilt about his behaviour or the use he is making of his symptoms, all point to a psychopathic personality. In the armed Forces one such individual may readily infect his weaker brethren, and the malingerer has been well compared with a typhoid carrier.

(2) *In hysteria*

Similarly, the hysterical patient is usually possessed of an unstable personality and immature emotional make-up. He is abnormally suggestible and often has rather poor mental equipment. His conflict, between the duty of doing something unpleasant and the desire to escape, results in a physical symptomatology which may closely mimic organic disease. A history of previous hysterical breakdowns, together with a history of similar illnesses in near relatives, emphasizes the underlying constitutional predisposition.

4. METHODS EMPLOYED

The choice of symptom by the malingerer, as by the hysteric, is often determined by emotional factors rather than by an intellectual process. The development of loss of function of a previously injured limb or eye is an example.

Numerous methods have been employed in malingering, and these tend to be employed more often in war-time, when instructors in malingering may make their appearance.

(1) *Production of skin lesions*

The skin is a readily available organ, and caustics, heat or other irritants may be used to produce lesions simulating skin diseases. Easily accessible areas are chosen, though rarely the face. The actual lesions are frequently linear with sharp margins. Aggravation of pre-existing eruptions may be practised when Workmen's Compensation is involved, as in industrial dermatitis.

(2) *Internal lesions*

The injection of paraffin under the skin has been used to produce tumour formation, and the subject may be thorough enough to produce a left-sided

Previous history

Accessible areas

supraclavicular mass as well as one in the epigastrium to simulate a carcinoma. Jaundice has been simulated by ingestion of picric acid, and albuminuria may be produced by taking cantharides, or by the more naïve method of adding egg-white to the urine. Mercury ointment, rubbed into the skin over the apex of the lung, has been known to produce an appearance on a skiagram of the lungs very similar to that of pulmonary tuberculosis.

(3) Feigned epilepsy

Feigned epilepsy is now a well-known method of attempting to avoid military service in war-time. Even tongue-biting and incontinence of urine can be faked, and the post-epileptic drowsiness simulated by administration of barbiturates before the doctor is hurriedly summoned. The description by witnesses of sudden loss of consciousness, falling and convulsion, completes the picture. When the subject is called up, soon afterwards, for military service, the doctor may, in all faith, give a certificate of epilepsy. Over-elaboration of the history, or an epidemic of similar cases, should point to the possibility of malingering. *Avoidance of military service*

(4) Blindness and deafness

Blindness or deafness may be feigned, or atropine may be instilled into the eye with resultant dilatation of the pupil and disturbance of vision. Irritation of the conjunctiva or lids by soap, tobacco ash or a caustic has been practised, to simulate trachoma.

(5) Weakness and sensory loss

Weakness of limbs or loss of sensation is a common presenting symptom in hysteria and may also occur in the malingerer. The weakness is unaccompanied by any wasting or reflex change, and glaring inconsistencies during the examination usually point to the psychogenic nature of the disability. The sensory loss does not fit in with anatomical concepts, varies during repeated examinations, and by suggestion can be made to wax or wane. It is frequently of the glove or stocking type, or limited to one-half of the body. *Inconsistency*

(6) Hyperthermia

Spurious hyperthermia

well and there are no accompanying signs of fever. By using two or more thermometers at the same time, or by testing the temperature of freshly voided urine, the ruse can usually be exposed. There is some doubt as to whether a true hysterical fever exists—that is to say, whether a hysterical patient can actually produce a true rise of body temperature.

5. DIAGNOSIS

The imitation of illness by the malingerer, as well as by the hysteric, depends not upon anatomical or physiological principles but on the subject's conception of the symptoms of the illness. The malingerer tends to over-act his part. He is too blind or too deaf. Over-elaboration, together with inconsistencies in the history and on examination, should rouse the examiner's suspicions. *Over-elaboration*

(1) Exclusion of physical disease

*Adequate
motive*

Diagnosis may, indeed, be very difficult. Recognition of the nature of feigned illness involves first of all the exclusion of structural or physical disease which might cause the symptoms. This is essentially a negative investigation and, in itself, does not provide sufficient evidence for a diagnosis of hysteria or malingering. As also in hysteria, the discovery of adequate motive is equally important. The clinical distinction between malingering and hysteria will depend largely upon the physician's impression of the patient's personality and self-knowledge.

The methods used in the clinical diagnosis of malingering and hysteria are very similar, the object being to "catch the patient out", as, for example, by the production of some glaring or bizarre inconsistency which transcends anatomical or physiological principles.

(2) Bizarre symptoms in organic disease

It should be emphasized, however, that bizarre symptoms may occur in organic disease, and that the latter may be present without signs on examination. Carcinoma of the pancreas, for instance, can result in severe pain without any sign of organic disease, and, if the patient has by nature a tendency to exhibitionism, he may be convicted of shamming. Addison's disease, with resultant general fatigue and pigmentation, may lead the unwary to mistake pigmentation for healthy looks and label the fatigue as functional. In myasthenia gravis, too, fatigue may be attributed to disinclination, and muscular weakness to hysteria. The behaviour of a patient with hypoglycaemia resulting from an overdose of insulin, or from an islet-cell tumour of the pancreas, may closely resemble that of a hysteric or a malingerer. Early nervous disease is particularly prone to give rise to difficulty in this connexion. The early symptoms of disseminated sclerosis may sometimes have a bizarre flavour to the inexperienced, and signs may be minimal. The general slowing-up of voluntary movement which occurs in early Parkinsonism without tremor, and the intracranial tumour which results in queer attacks and odd behaviour, without headache or papilloedema, are particularly likely to be diagnosed as hysteria or malingering.

6. ASSOCIATION WITH ORGANIC DISEASE

*Exaggeration
of symptoms*

It is common clinical experience, especially in nervous disorders, to discover organic disease with a so-called functional overlay or veneer, and one of the difficult problems constantly confronting the clinician is the separation of these two aspects in a given illness. Hysterical symptoms may occur in a person with undiagnosed organic disease, if the patient receives insufficient attention for his organic symptoms, or if he is constantly told that there is nothing wrong. He may be forced to add to the clinical picture or exaggerate his symptoms. Hysteria, and possibly even malingering, may therefore be associated with, and even mask, organic disease, and it is possible, too, that malingering may develop into hysteria, the patient finally deceiving himself.

7. TREATMENT

From the very nature of his behaviour the malingerer is psychopathic, and any attempt to score a personal triumph over the patient should be avoided.

Care should be taken with regard to accusations of dishonesty. After exposure of the simulation, further inquiry into the motive is indicated and psychotherapy may be advisable.

BIBLIOGRAPHY

Buñewitsch, K. (1941). *Wien. med. Wschr.*, 89, 472.

Good, R. (1942). *Brit. med. J.*, 2, 359.

Hurst, A. (1944). *Medical Diseases of War*, 4th ed., p. 19. London; Arnold.

Ironside, R. (1940). *Brit. med. J.*, 1, 703.

MacNeal, W. J. (1939). *Arch. Intern. Med.*, 64, 800.

Norris, D. C. (1943). *Practitioner*, 150, 363.

[References to other titles are given under *Malingering*, in the Index Volume. The subject is also dealt with in the *British Encyclopaedia of Medical Practice* (1938) Vol. 8, p. 354.]

MANIPULATIVE SURGERY

By W. D. COLTART, F.R.C.S.

ASSISTANT ORTHOPAEDIC SURGEON, ST. BARTHOLOMEW'S HOSPITAL, LONDON

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PART I

GENERAL CONSIDERATIONS

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229.] Manipulation is as old as surgery itself, and has been used continuously by orthopaedic surgeons and others for many years. The bone-setters and the osteopaths have established their reputations by the manipulation of joints, but it is not necessary to accept the osteopathic theories of disease to understand the success of their methods of treatment. There is no doubt that, in properly selected cases, the combination of an expert manipulative technique with careful attention to post-operative exercise therapy will produce successful cures whether this method is applied by the qualified or the unqualified practitioner.

1. RATIONALE OF MANIPULATION

There is no need to assume that manipulation succeeds only because something which was out of place has been replaced, especially when these displacements cannot be demonstrated by clinical examination or by radiography. Manipulation is nothing more than passive movement. The joints are moved, and through movement traction is applied to synovial membrane, capsule, ligaments, muscles or tendons. If any of these structures is adherent or contracted, the manipulation may succeed in freeing or stretching the affected tissue.

Contracture and adherence of soft tissues are well-recognized sequelae of many traumatic, toxic and inflammatory processes, and their pathology and morbid anatomy are known only too well. Contracture and adherence can be recognized clinically and are sometimes so marked that there is gross

limitation of movement or even fibrous ankylosis of the joint; there may be actual distortion of the limb, such as an equinus deformity from contracture of the muscles of the calf. In this type of case, manipulation is unlikely to be of great value.

In milder conditions there may be limitation of the range of movement of a joint in but a single direction—for example, the final degrees of flexion at the knee; passive attempts to force full flexion are painful. In such cases the patellar tendon, or the anterior part of the capsule of the knee joint, is contracted or adherent, and it is the diminished mobility of these structures which is preventing flexion of the joint.

Limitation of movement in one direction

In another group of cases it may be observed that although the range of movement at the joint is not apparently limited, yet movement in one direction is painful. It may be supposed that although adherence or contracture in capsule, ligament, muscle or tendon is not sufficient to limit movement, mobility is retained only at the price of stretching adherent or contracted tissues. In these cases, too, it is found that forcible movement of the joint through its full range can relieve pain.

Painful movement in one direction

There are certain regions in the body in which a syndrome occurs, characterized by acute pain of sudden onset and associated with fixation and deformity. The knee and the temporo-mandibular joint are each subject to sudden attacks of this nature, which we call locking. Explanation of the locking of knee and temporo-mandibular joints is simple. Both possess intra-articular fibrocartilages, and displacements of these cartilages are a proven cause of locking. This internal derangement in either joint can be relieved by an appropriate manipulation.

Syndrome of locking

Locking with intra-articular cartilage

In other situations, however, when there is no intra-articular cartilage, the syndrome is not readily explained. Relief may be sudden, sometimes after a movement in a certain direction, or in performing some simple task. On the other hand, the acute features may subside gradually during the ensuing hours or days. Whether this relief of acute pain is sudden or gradual, it is often followed by chronic painful symptoms of variable duration. The acute attacks have often been completely relieved after manipulation, and osteopaths have argued backwards from their cures by manipulation and have postulated that a bone was out of place.

Locking without intra-articular cartilage

The common situations in which this syndrome appears are the neck (acute torticollis), and the lower back, where the symptoms may be referred to the lumbo-sacral area or to one or other sacro-iliac area and may be associated with acute pain in the distribution of the sciatic nerve. Somewhat similar attacks may affect the shoulder but in this situation the characteristic element of deformity is lacking, although there may be intense muscle spasm, and sudden relief spontaneously or by manipulation is unusual. That the fixation and deformity which occur in the neck (torticollis) and lower back (scoliosis, kyphosis or flattening of the lumbar curve) are consequent on muscular action appears certain, because when the patient is fully relaxed under an anaesthetic both deformity and fixation disappear. Unfortunately, both may recur when consciousness is regained. The cause of the muscle spasm requires further consideration. Trauma and infection are obvious possibilities but it is difficult to understand how spasm brought about by rupture of a few fascial or muscle fibres, or by a fibro-myositis, can be

Common situations

Characteristic deformity

relieved with such dramatic suddenness by manipulation. The rupture of the ligamentous part of an intervertebral disc followed by protrusion of the nucleus pulposus is undoubtedly the cause of the acute onset of symptoms in many cases of sciatica. Can this be the explanation in all cases of this characteristic syndrome when it affects the lumbo-sacral or sacro-iliac area rather than the distribution of the sciatic nerve, and is the rupture of an intervertebral disc in the cervical region the usual cause of an acute torticollis of this type? It may be that this is the explanation, but even so it is not possible to claim that cure by manipulation indicates that a protruding disc has been replaced.

2. INDICATIONS FOR MANIPULATIVE SURGERY

Manipulative surgery may be indicated in the treatment of fractures and dislocations, for the correction of a deformity, for restoring mobility to joints and for the relief of painful symptoms in certain conditions.

This article is not concerned with methods used in the reduction of fractures and dislocations, or with the manipulative correction of deformity.

(1) Manipulation for restoring mobility

The morbid changes which limit joint mobility may be the result of an injury, or of an acute, subacute or chronic arthritis. In addition, a joint may become suddenly locked either because of the presence of a loose body in the joint or because of the displacement of an intra-articular fibrocartilage. Manipulation will not directly overcome muscular spasm, nor will it affect irregularity of the bony surfaces, but in these other conditions in which movement is limited by contracture, or adhesions, or locking, it may be extremely valuable.

The results of manipulation are disappointing when stiffness is of long duration, or when the limitation in range is considerable or is part of a chronic arthritis of the hypertrophic or atrophic type.

If the clinical condition suggests that it should be possible to restore or improve the range of movement, manipulation is most usefully employed in combination with exercise therapy. To restore movement by forceful manipulation and not to train the patient to follow up this improvement by exercises, is to run a great risk of recurrence of, if not an actual increase in stiffness.

In mobilizing a joint in which movement is limited, manipulation is used in conjunction with exercises: (a) to start off movement which is proving difficult and resistant to exercise alone; (b) when treatment with exercise alone has come to a halt, and (c) to speed up restitution of the last few degrees of movement during a successful course of exercises.

The treatment of joints which become locked because of a loose body or displaced fibrocartilage does not require special consideration, except to say that failure to unlock the joint by a properly conducted manipulation is usually a final indication for arthrotomy.

(2) Manipulation for the relief of pain

There are cases in which painful symptoms can be reproduced by the passive stretchings of certain structures. Common examples are the external lateral

*Loose body
Displacement
of intra-
articular
fibrocartilage*

*Manipulation
in conjunction
with exercises*

ligament of the ankle, the tarsal ligaments, the internal lateral ligament of the knee, the dorsal carpal ligament, and the attachment of the extensor muscles of the wrist and fingers to the lateral epicondyle of the humerus. In some cases, pain is accompanied by actual limitation of movement in the direction which stretches the particular structure involved. In others, although movement is painful it is not limited. The characteristic feature is pain on movement of the joint in one direction only. Sometimes the movement which causes pain is one not normally under the voluntary control of the individual an—for example, abduction of the tibia on the femur, which may cause pain in adherent internal lateral ligament of the knee.

Pain on movement in one direction

These painful conditions can often be cleared up by a manipulation which stretches the affected structure. It is well to encourage the patient to follow up the manipulation with his own exercises, but when pain rather than limited movement is the main feature, after-treatment by exercise is not so essential. It is in this type of case that the "miraculous" cures of the bone-setters and osteopaths are achieved by a single, powerful but skilfully applied "wrench".

There is another group of cases in which pain occurs because, owing to some defect of normal posture, certain ligaments are overstretched. In the course of time, the stretched ligaments become painful. This type of pain most frequently occurs in the back and in the foot; in either of these situations there may be evidence of a postural defect in the shape of an actual deformity, such as round shoulders, exaggerated lumbar lordosis or flat feet. Nevertheless, chronic overstretching of the ligaments can occur without deformity when the defect in the postural mechanism is a loss of tone in those muscles which would normally protect the ligaments from strain. Manipulation is useful in relieving this type of pain but is only part of a regimen of treatment which will include exercises to correct the abnormal posture and re-educate and strengthen muscles which are not playing their part in the postural mechanism.

Defective posture

Pain in the neck or in the lower part of the back, of sudden onset and associated with fixation and deformity, can sometimes be relieved in a dramatic manner by manipulation. It must be borne in mind, however, that the syndrome may be due to the rupture of an intervertebral disc, and manipulation must be undertaken with extreme caution, because cases have been reported in which manipulation has produced further displacement of the nucleus pulposus, with damage to the spinal cord or cauda equina.

Risk of damage to spinal cord

3. MANAGEMENT OF CASES FOR MANIPULATION

(1) Diagnosis

Careful clinical examination should be supplemented by radiography before any manipulation is undertaken. The affected joint may be tuberculous; there may be a recent unsuspected fracture or a fracture in which union is not secure; there may be a secondary neoplastic deposit in a vertebral body; pain in the knee may be referred from a diseased hip joint, or a skiagram may reveal an unsuspected bony irregularity in the joint which is the cause of loss of movement and cannot be affected by manipulation.

Radiography essential

The erythrocyte sedimentation rate may provide a useful guide to the activity of a chronic arthritis and, therefore, to the prognosis of manipulation, but it is not infallible.

Erythrocyte sedimentation rate

(2) General rules for manipulation

The following points should be observed in manipulative treatment.

"Ideal case"

(1) The "ideal case" for manipulation is one which exhibits pain on movement in a single direction, especially if the movement in that direction is a little limited.

(2) Manipulation should be preceded by a course of mobilizing exercises; these are principally to teach the patient what to do after manipulation, but the symptoms may clear up without it.

(3) When manipulating, it is as well to grasp the limb as near to the joint as possible, so that the lever used is short. The manipulation must be performed with strength to be effective, and fractures can easily occur.

(4) As well as ordinary movements, those which are not normally under voluntary control should be carried out. These are:

(a) separation of the joint surfaces by traction (distraction);

(b) lateral displacement, inwards and outwards, as well as abduction and adduction;

(c) displacement forwards and backwards (rocking); and

(d) rotation.

(5) A distinction should be made between the manipulation which is for the relief of pain and the manipulation undertaken to restore movement. If the former, it is usually advisable and possible to put the joint through its full range of movement in all directions. If the latter, too much should not be attempted at one sitting and several manipulations should be planned, with intervening courses of exercise.

(6) The muscles must be fully relaxed at the time of the manipulation. Osteopaths and bone-setters achieve this by a skilled manual and conversational technique. The majority of surgeons use an anaesthetic.

(3) Anaesthesia

Some form of anaesthetic is used almost invariably. The objectives are to secure adequate relaxation and to render the procedure painless, in that order. For manipulation of the foot, wrist, elbow and sometimes for the knee, properly administered nitrous oxide will suffice. For adequate manipulation of the back, shoulder, hip, neck and temporo-mandibular joints, an anaesthetic which will produce relaxation for a longer period is required: intravenous soluble thiopentone (Pentothal Sodium) is very satisfactory.

Nitrous oxide

Pentothal Sodium

(4) After-treatment

Reaction

There is sometimes a painful reaction after manipulation, but this should be unusual. If pain does occur, it should be relieved after a short time by rest and analgesics. A serious degree of pain or swelling after manipulation suggests that the procedure has been overdone, or that the case was unsuitable.

Physiotherapy

Physiotherapy is an important accessory to manipulation. Exercise forms the basis of treatment, and it is advisable that the patient should meet his physiotherapist and learn simple exercises before the actual manipulation is carried out. He should be convinced of the importance of exercise and should not be allowed to place his confidence in massage and electricity. Exercises are resumed on the day after the manipulation, and the patient should carry them

out regularly himself, although he may visit, or be visited by, the physio-therapist only three or four times a week.

(5) Dangers of manipulation

(a) *Improper selection of cases*

The danger of improper selection of cases should be avoided if proper care is taken over diagnosis.

(b) *Trauma of manipulation*

The accidents which may occur in the hands of the inexperienced include the following: fractures of the patella; avulsion of the tibial tubercle; fracture of the upper end of the humerus; fracture of a metatarsal bone; crush fracture of the body of a vertebrae, and retropulsion of an intervertebral disc.

Fractures are apt to occur if the bones are atrophic from immobilization or senility, or if the manipulation is carried out when the patient's muscles are not adequately relaxed.

(6) Failure in manipulation

Manipulation may fail to effect improvement in a range of movement, or to relieve painful symptoms, in the following circumstances: (a) if the case has not been properly selected; (b) if too much force is used, so that an excessive reaction is followed by increased stiffness; (c) if too little force is used; (d) if muscle relaxation is inadequate, so that some of the force required has to be used in overcoming the resistance of the patient's muscles; (e) if the movements are not carried out in the right directions to overcome the particular contracture or adherence of the soft tissues; (f) if those movements which are not under voluntary control, such as distraction or rocking, are omitted; and (g) if preliminary and post-operative exercises are forgotten.

PART II

INDICATIONS FOR, AND TECHNIQUE OF, MANIPULATION OF INDIVIDUAL JOINTS AND REGIONS

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1. THE ANKLE AND FOOT

(1) Indications

(a) Chronic sprain of the ankle

Adherence or contracture of the capsule of the ankle joint is common after injuries and after arthritis. In many of these cases, when the limitation of mobility is slight, painful symptoms may be cured by manipulation.

The symptoms may be localized to the outer side of the joint and it may be found that the anterior fasciculus of the fibular collateral ligament is tender and that pain occurs when the foot is inverted and plantar flexed so that the ligament is stretched. The condition is known as chronic strain of the ligament and responds well to manipulation.

Both chronic sprain of the ankle and chronic strain of the fibular collateral ligament may be the result of faulty walking, or the wearing of high-heeled shoes.

Symptoms

Chronic strain

(b) Chronic foot strain

Painful symptoms in the foot may occur from overstretching of the tarsal ligaments as a result of muscular incompetence alone, or may be associated with actual deformity. On occasions, foot strain manifests itself acutely; in such cases there is no treatment so effective as rest followed by graduated exercises, but in the milder cases for which the title chronic is appropriate, manipulative treatment is indicated. It should be remembered that a chronic foot strain does not occur only in the person with flat foot. Indeed, symptoms are more common in those with a foot of normal shape, in those with a high-arch foot and in those with all types of club foot.

(c) Tarsal osteoarthritis

Mild degrees of painful limitation of movement can be relieved by manipulation.

(d) Metatarsalgia

True Morton's disease is now recognized as being due to a localized fibrosis *Morton's disease* of an interdigital nerve and manipulation is not indicated. Many patients complain of chronic pain in the metatarsal region, and passive stretching of the capsules of the metatarso-phalangeal joints is painful; this type of case often responds to manipulation.

(e) Hallux rigidus

In a fully developed case of hallux rigidus, the metatarso-phalangeal joint of the great toe is extremely stiff and there may be well-marked radiographic signs of osteoarthritis. This type of case is unsuitable for treatment by manipulation, but a chronic sprain of the joint may appear as the result of an injury and symptoms will be relieved by manipulation and exercises, particularly in adolescents.

(2) Technique of manipulation*(a) Combined manipulation of the tarsus and ankle*

The technique of this procedure involves the following four movements which are carried out on each foot and produce a combined effect on the tarsus and ankle: (i) inversion and adduction; (ii) eversion and abduction; (iii) plantar flexion, and (iv) dorsiflexion.

The patient lies on his back, with both legs exposed as far as the knees, and with the feet at the end of the table. The surgeon stands at the end of the operation table, facing the patient's feet; the assistant grasps and steadies the knee. The following description applies to manipulation of the left foot; the manipulation of the right foot is similar, except that the position of the operator's hands is reversed in each instance.

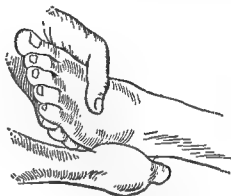
The heel is rested in the surgeon's left palm, and grasped with the fingers and thumb, the thenar eminence lying to the inner side of the heel and acting as a fulcrum for movement at the subastragaloid and mid-tarsal joints. The surgeon's right hand grasps the dorsum of the foot at the level of the bases of the metatarsal bones, his thumb lying across the plantar surface of the foot. If the foot is held too near the toes, and is squeezed rather than held firmly, a fractured metatarsal bone may result.

(i) *Inversion and adduction* (Fig. 1 (a)).—The foot is swung into full inversion and is adducted at the mid-tarsal joint; the surgeon's left hand remains steady, and his body-weight is transferred from his right to his left foot.

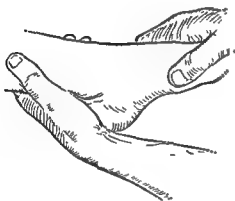
(ii) *Eversion and abduction* (Fig. 1 (b)).—The surgeon changes the position of his hands. The left hand grasps the bases of the metatarsal bones from the



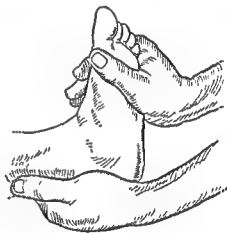
(a) Inversion and adduction.



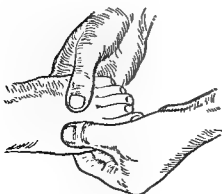
(b) Eversion and abduction.



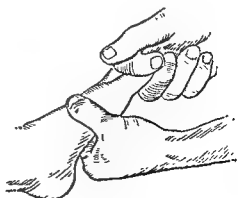
(c) Plantar flexion.



(d) Dorsiflexion



(e) Moving adjacent metatarsal bones



(f) Manipulation of metatarso-phalangeal joint.

FIG. 1.—Manipulation of the foot, ankle and toes

inner side, the thumb lying on the dorsum of the foot. The surgeon's right hand fixes the heel, the thenar eminence being on the outer side. The foot is then swung into full eversion, the surgeon's weight being transferred in the opposite direction from his left to his right foot.

(iii) *Plantar flexion* (Fig. 1 (c)).—Plantar flexion at the ankle joint is combined with inversion and supination of the foot. The surgeon's left hand again steadies the heel but allows it to move upward, and his right hand is placed across the dorsum of the foot forcing full plantar flexion and almost full inversion.

(iv) *Dorsiflexion* (Fig. 1 (d)).—The surgeon's left hand grasps the leg immediately above the ankle, the palm of his right hand is applied to the anterior part of the sole of the foot and, with his right elbow acutely bent, the surgeon applies his body-weight to dorsiflex the patient's foot.

Manipulation of metatarsal bones and toes is performed at the same time.

(b) *Manipulation of the metatarsal region and toes*

Manipulation of the metatarsal region and of the toes is carried out in two parts: (i) movements of metatarsal bones upon each other; (ii) manipulation of the metatarso-phalangeal and interphalangeal joints.

(i) *Movement of metatarsal bones upon each other* (Fig. 1 (e)).—Two adjacent metatarsal bones are gripped in their distal halves by the surgeon's finger and thumb of each hand. The metatarsal bones are then moved, the one upward, the other downward towards the sole of the foot; the movement of each bone is then reversed.

(ii) *Manipulation of the toes* (Fig. 1 (f)).—Each toe is held in turn by the surgeon's right hand, his left hand fixing the metatarsal region of the foot. The toe is pulled in its long axis away from the corresponding metatarsal head and is rapidly moved into flexion, dorsiflexion, abduction and adduction and also rotated. Extension and flexion at the interphalangeal joints are then carried out through as full a range of movement as is possible, a steady extending force in the long axis of the toe being applied throughout.

The description of these manoeuvres occupies considerably more time than their actual performance, and a complete manipulation of both feet, including the metatarsal region and toes, can be carried out under nitrous oxide anaesthesia.

2. THE KNEE

(1) *Indications*

In considering treatment by manipulation it is obviously important to distinguish between those cases in which the disability of the knee is directly attributable to locking and those in which it is due to contracture or adherence of ligaments or capsule. The purpose of the manipulation is quite different in the two groups.

(a) *Locked knee*

One of the manifestations of internal derangement of the knee is locking. This may be due to a torn and displaced meniscus, a fracture of the spine of the tibia, or a loose body. In addition there are many conditions which will produce sudden muscular spasm which inhibits movement in the knee and may simulate a true lock. A skiagram will reveal a fracture and most loose

bodies, and examination of the joint under an anaesthetic will eliminate muscle spasm and distinguish the pseudo-lock from the true lock. When the patient is under an anaesthetic it may be possible at the same time to release by manipulation a knee which is truly locked.

(b) Adherence of ligaments

An acute strain of the tibial collateral ligament often simulates a lesion of the medial meniscus and the latter may, indeed, be a concomitant injury. As the result of trauma the ligament may become adherent, and flexion of the knee—a movement which stretches an adherent medial collateral ligament—becomes painful; similarly ordinary walking and standing cause pain because of the abducting strain on the ligament. Sudden overstretching of this ligament, which may occur when walking or running on uneven ground, produces attacks of pain which again cause confusion in the diagnosis from a torn internal cartilage. The lesion can be recognized by localization of pain and tenderness to the medial ligament and by pain produced when the ligament is put on the stretch by passive abduction of the tibia on the femur. Manipulation produces a cure provided that the ligament has not been actually torn or grossly overstretched and that there has been no bone formation in its fibres (Pellegrini-Stieda lesion).

Pellegrini-Stieda lesion

(c) Contracture of the capsule

As a result of severe injury, infection, or chronic arthritis there may be gross limitation of flexion or extension of the knee. This type of fibrosis responds badly to manipulation, but a lesser amount of contracture may limit the last few degrees of flexion or extension, and both pain and interference with movement can be relieved by manipulation.

(2) Technique of manipulation

(a) Manipulation of the locked knee

Unless the patient is seen immediately after the accident, manipulation for locking of the knee joint is best performed under anaesthesia. Nitrous oxide gas skilfully administered is often sufficient, but preliminary preparations for deeper anaesthesia should be made.

Gas anaesthesia may be adequate

The surgeon, having satisfied himself that the patella is mobile and not dislocated outward, flexes the knee fully and in this position he rotates it in either direction. This is often the effective part of the manoeuvre for cartilage lesions, but it is customarily followed by what is known as the classical method of unlocking the knee and obtaining full extension. With one hand the surgeon grasps the thigh, and with his other hand he grips the ankle and foot, and abducts and externally rotates the tibia upon the femur (Fig. 2 (c)). The knee is then extended by its own weight, guided by the hand on the thigh, whilst the tibia is held in position by the other hand. The knee is held in this position for a few moments, and is then allowed to straighten with external rotation and persistent adduction. If these methods fail, the remainder of the routine movements described below are carried out. A knee locked by reason of a loose body in the joint may be freed by any combination of the routine procedures.

Cartilage derangements

Success, which may be heralded by an audible click, is indicated by the restoration of full extension. If the operator is at all uncertain whether this

has been achieved the patient will be able to remove all doubts after he has recovered from the anaesthetic. A compression bandage should always be applied after manipulation of the locked knee.

(b) Routine manipulation

The following movements are carried out in routine manipulation of the knee. (i) Lateral movement of the patella. (ii) Flexion of the knee. (iii) Rotation.

(iv) Antero-posterior movement. (v) Extension.

The surgeon stands to the outer side of the affected limb, the patient lying supine.

(i) *Lateral movement of the patella* (Fig. 2 (a)).—This is the first procedure. A fracture of the patella may result so easily from flexion that it is *Danger of flexion* essential to make certain that the patella is not adherent.



(a) Lateral movement of the patella.



(b) Flexion of the knee.



(c) External rotation



(d) Antero-posterior movement



(e) Extension.

FIG. 2 —Manipulation of the knee

In addition, freeing the patella may allow a few more degrees of flexion at the knee joint. The patella is grasped by the fingers and thumb of each hand, one at its upper, the other at its lower pole, and it is moved laterally and medially to its fullest extent. A little upward and downward movement can sometimes be obtained.

(ii) *Flexion of the knee* (Fig. 2 (b)).—The surgeon with one hand grasps the thigh just above the adductor tubercle of the femur. With his other hand he grasps the leg in its middle third and flexes the knee slowly. The amount of force exerted in attempting to restore flexion must depend upon the type of case. If the quadriceps muscle is fibrosed or if the patella is adherent, forcible flexion may result in fracture of the patella, separation of the tibial tubercle, or rupture of some other part of the extensor apparatus, or even in fracture of the femur.

(iii) *Rotation* (Fig. 2 (c)).—With the knee flexed, the grip of the surgeon's right hand is transferred to the ankle and foot; the leg is rotated outward and inward several times, the knee being gradually allowed to extend meanwhile.

(iv) *Antero-posterior movement* (Fig. 2 (d)).—The knee is flexed to a right angle. The patient's thigh is supported by an assistant, or the leg can be allowed to hang over the end of the table. The leg is then grasped by the surgeon with both hands, in its upper third, and rapid backward and forward movements of the tibia on the femur are carried out.

(v) *Extension*.—Provided the knee is not locked the method shown in Fig. 2 (e) may be used for restoring the last few degrees of extension. The patient's heel is placed across the shoulder of the surgeon, who clasps his hands over the front of the knee and pulls it towards his chest. Only moderate force should be used.

3. THE HIP

(1) Indications

(a) *Osteoarthritis*

Manipulation is occasionally successful in relieving pain in comparatively early cases of osteoarthritis in which mobility of the joint is well preserved but movement in one direction is painful.

(b) *Capsular strain associated with deformities*

Such conditions as coxa plana, congenital subluxation and congenital dislocation often give rise to chronic pain during adult life. Although there is usually a secondary osteoarthritis, on occasions the pain is due to overstretching of ligaments and can be relieved by manipulation. The procedure is well worth a trial in the treatment of these disabilities.

(c) *Chronic sprain*

Sprains of the hip are rare but do occur. Manipulation is successful in the relief of pain and in regaining full movement when capsule and ligaments have become adherent.

(2) Technique of manipulation

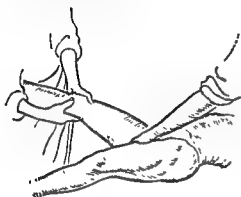
The movements carried out in manipulating the hip are as follows.

- (i) Distraction. (ii) Flexion. (iii) Rotation. (iv) Abduction and adduction.
- (v) Extension.

The patient lies supine. The operator stands at the patient's affected side with an assistant standing opposite.

(i) *Distraction* (Fig. 3 (a)).—Manual traction is applied to the limb, against the counter-traction applied by means of a towel or the assistant's hand placed in the groin.

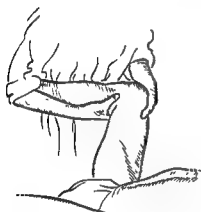
(ii) *Flexion* (Fig. 3 (b)).—This movement is performed with the knee flexed.



(a) Distraction and abduction.



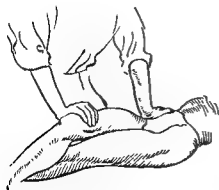
(b) Flexion.



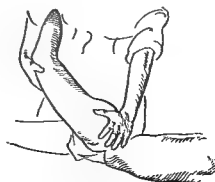
(c) External rotation.



(d) Internal rotation.



(e) Adduction



(f) Extension.

FIG. 3—Manipulation of the hip

(iii) *Rotation* (Fig. 3 (c) and (d)).—This is carried out with the hip flexed. It is important to avoid extension in performing rotatory movements, as this would endanger the neck of the femur.

(iv) *Abduction and adduction* (Fig. 3 (a) and (e)).—During these movements the assistant fixes the patient's pelvis on the affected side with one hand and the unaffected lower limb with his other hand. The movements may be combined with traction.

(v) *Extension* (Fig. 3 (f)).—The patient is rolled on to the unaffected side or into the prone position. For the right hip, the operator places his left hand on the gluteal region above the hip joint and grips the patient's thigh above the knee with his right hand. The hip joint is then extended by pulling backward with the right hand; counter-pressure is exerted by the left hand. The range of movement of individual hip joints varies considerably, so that in this manipulation it is usual to compare the range obtained with that in the unaffected joint.

4. THE NECK

(1) Indications

(a) *Sprains of cervical joints*

The intervertebral joints are liable to injuries and sprains in the same way as is any other joint. Chronic pain may follow an injury to the neck and can be relieved by manipulation.

(b) *Cervical osteoarthritis*

Few cases are suitable for manipulation but, as in other sites, the occasional case in which movement appears to be limited and painful in one direction only can be relieved.

(c) *Cervical fibrositis*

The trapezius muscles are often affected by fibro-myositis. The clinical condition is well recognized and is often extremely resistant to treatment. A single manipulation under an anaesthetic often proves a useful adjuvant to physiotherapy in the later stages.

(d) *Acute torticollis*

Acute, painful wry neck is a familiar event, and has already been referred to in discussing the syndrome of sudden fixation in various regions of the body. Manipulation of the neck may bring immediate relief, but the pain and

deformity may be due to subluxation or dislocation at the atlanto-axial joint, or to retro-pulsion of an intervertebral disc. There must be careful radiological investigation and due consideration of all the features of the case before resorting to manipulation.

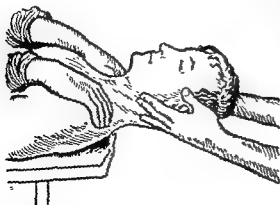


FIG. 4.—Manipulation of the neck: distraction.

(2) Technique of manipulation

The patient lies on his back with his shoulders at the end of

the table. The surgeon sits at the end of the table and supports the head in his hands (Fig. 4); the surgeon's fingers are hooked round the angle of the jaw and the palms support the side of the occiput. An assistant, facing the operator, fixes the shoulders. The movements are as follows.

(i) Distraction. This should be carried out by a steady pull, which is maintained while the following movements are performed. (ii) Forward flexion. (iii) Lateral flexion. (iv) Extension. (v) Rotation.

All these movements are performed slowly and without jerking. It is unnecessary to alter the position of the hands supporting the head.

5. THE BACK

(1) Indications

(a) *Injuries*

Injuries of all grades of severity to the back are extremely common and chronic backache is a notorious sequel which may prove disabling and difficult to cure. If the pain is due to capsular, ligamentous or muscular contracture or adherence it should, theoretically, respond to manipulation and exercises. The back is a composite structure and the number of joints and muscles affected may be considerable. Furthermore, the anatomy of the joints and muscles makes it difficult to apply a local stretching force to any particular area; consequently exercises or manipulation may not, as it were, reach the affected structure. *Chronic backache*

(b) *Chronic strains of postural origin*

Individuals with deformities of the spine may at some stage complain of chronic backache, and it is assumed that this pain is consequent upon chronic overstretching of ligaments or muscles. Backache may have the same causation when there is no deformity but when the muscles fail to protect the ligaments from strain. This may occur under conditions of ill health, overtiredness, obesity or bad posture. Backache from chronic strains of this nature may prove as difficult to cure by manipulation and exercise as the backache which occurs after an injury, and for the same reasons. *Deformities of the spine*

Manipulation of the back under an anaesthetic has, however, become an accepted and routine procedure in the treatment of chronic backache which does not respond to exercises alone; success will be gained in a number of cases.

It must be emphasized that manipulation of the back should not be undertaken without a most thorough clinical and radiological survey of the patient.

(c) *Protrusion of an intervertebral disc*

This is not the place to describe the pathology and clinical features of a prolapsed or retropulsed disc and the surgery of this condition will be described in another volume. Manipulation may sometimes bring about dramatic improvement but there is some danger of causing a further protrusion and an aggravation of the symptoms, or even of damaging the spinal cord. These dangers may be minimized if neither forced flexion nor extension be employed. The symptoms associated with a ruptured intervertebral disc, whether backache alone or backache and sciatic pain, tend to subside with rest, modification of activity and the lapse of time. Manipulation is, therefore, reserved *Danger of damage to spinal cord*

for those cases in which a reasonable trial of conservative treatment has failed to bring relief and for those cases in which economic or similar considerations forbid delay.

(2) Technique of manipulation

Routine method

The routine method of "manipulation of the spine" involves the lumbo-sacral and sacro-iliac joints in addition to the thoracic and lumbar regions of the spine.

The manipulation consists in the following movements. (i) Flexion of each sacro-iliac joint. (ii) Forward flexion of the spine. (iii) Lateral flexion from the position of forward flexion. (iv) Rotation of the spine and distraction of the sacro-iliac joints. (v) Extension of the spine.

(i) *Flexion of each sacro-iliac joint* (Fig. 5 (a)).—With the patient in the supine position and the opposite limb held on the couch by the assistant, the surgeon flexes the hip of the side under treatment, keeping the knee extended. This position may be maintained by the surgeon applying his shoulder to the back of the patient's calf and clasping his hands in front of the knee. Flexion is then gently forced by a cautious forward rocking of the operator's trunk.

Necessity for assistance

(ii) *Forward flexion of the spine* (Fig. 5 (b)).—The patient lies in the supine position. The operator, standing on the right side of the patient, places his right forearm beneath the patient's knees. Flexion of the spine is then carried out by lifting the knees forward and upward until they are in the same vertical plane as the patient's chin. The operator will sometimes require assistance in this movement if the patient is heavy or unwieldy; if this is so, the assistant lifts the patient's buttocks and helps in supporting the weight, but he should not be allowed to take any part in the actual flexing of the spine. The application of the operator's full body-weight has been advocated, but this must be condemned as being both dangerous and unnecessary. Unduly vigorous flexion of the spine in the relaxed patient transmits a very great compression force to the vertebral bodies and intervertebral discs, and two serious complications caused by therapeutic manipulation have occurred, each more than once; these complications are crush fracture of the body of a normal vertebra and damage to the cauda equina from displacement of part of a ruptured disc. It should be performed with the least possible force.

Dangers of too vigorous flexion

With the patient in the supine position, the operator stands on the right side of the patient, places his right forearm beneath the patient's knees, and lifts the knees forward and upward until they are in the same vertical plane as the patient's chin. The operator will sometimes require assistance in this movement if the patient is heavy or unwieldy; if this is so, the assistant lifts the patient's buttocks and helps in supporting the weight, but he should not be allowed to take any part in the actual flexing of the spine. The application of the operator's full body-weight has been advocated, but this must be condemned as being both dangerous and unnecessary. Unduly vigorous flexion of the spine in the relaxed patient transmits a very great compression force to the vertebral bodies and intervertebral discs, and two serious complications caused by therapeutic manipulation have occurred, each more than once; these complications are crush fracture of the body of a normal vertebra and damage to the cauda equina from displacement of part of a ruptured disc. It should be performed with the least possible force.

flexion; the thorax is fixed by an assistant placing a hand on each side of the chest.

(iv) *Rotation of the spine and distraction of the sacro-iliac joints* (Fig. 5 (d)).—For this movement, the patient is placed on to his side, first on to the right side. The surgeon stands opposite the patient's abdomen. The patient's left arm hangs behind him and away from the surgeon; the left lower limb hangs over the edge of the table towards the surgeon. This produces a twist in the long axis of the spine. The surgeon places his left hand on the left shoulder, and his right elbow and forearm on the crest of the uppermost—in this case the left—ilium. The assistant steadies the patient's right leg, and the right leg only. The operator rotates the spine and distracts the left sacro-iliac joint in

one vigorous movement, by pulling the ilium towards him and pushing the shoulder away. The effectual performance of this movement requires practice. The contralateral joint is similarly manipulated with the positions reversed, the patient lying on the left side, and the surgeon going to the other side of the table.

(v) *Extension of the spine* (Fig. 5 (e)).—The patient is turned face downward. The surgeon stands at the patient's left side and passes his right arm in front of the thighs. He lifts the thighs vertically, so that the pelvis is raised, and only the head, shoulders and chest remain upon the table. Hyperextension is secured by short, carefully moderated thrusts with the surgeon's left hand placed at various points in the lumbar and lumbo-sacral regions.



(a) Flexion of the right sacro-iliac joint.



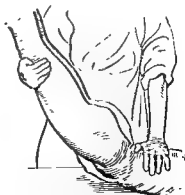
(b) Flexion of the spine



(c) Lateral flexion of the spine from the position of forward flexion



(d) Rotation of the spine and distraction of the left sacro-iliac joint.



(e) Extension of the spine.

FIG 5 —Manipulation of the spine.

6. THE SHOULDER

(1) Indications

(a) Chronic arthritis of the acromio-clavicular joint

There are several morbid conditions which will give rise to chronic pain in the shoulder. Amongst them, the symptoms of chronic sprain and of osteoarthritis of the acromio-clavicular joint may often be cured by a manipulation of the shoulder which puts the joint through a full range of movement. Manipulation is not indicated in the treatment of subacromial bursitis or of the various lesions of the supraspinatus tendon, except for the relief of stiffness of the shoulder from an associated peri-arthritis.

(b) Peri-arthritis of the shoulder joint

Stiffness of the shoulder is common after fractures and other injuries, and manipulation, as part of a regimen of exercises, is of assistance in regaining mobility. In manipulating a shoulder there is a special risk of doing too much at one session and thereby producing a severe and adverse reaction. The risk is less if manipulation is used when improvement from mobilizing exercises has come to a halt, or if it is used to regain the last few degrees of movement after a successful course of exercise, than when it is used to begin movement in a joint which is very stiff. Nevertheless, manipulation is of considerable use in starting movement of a stiff joint. In practice it is usually found that when the patient is fully relaxed under a general anaesthetic the first attempts at external rotation and abduction produce an immediate increase in range, often with a tearing sensation. After this it may be possible to move the shoulder through its full range in all directions with ease and without the need for any further force. In this event it is obviously unnecessary to do more and a successful result can be expected. On the other hand, further movement may be absolutely obstructed and it would be unwise to proceed further at that session, although another manipulation may be indicated in a few weeks, and after a further course of mobilizing exercises has been carried out.

A particular form of peri-arthritis occurs spontaneously in middle-aged and elderly people and is known appropriately enough as "frozen shoulder". Peri-arthritis of this nature is extremely painful. Manipulation is of no value in the early stages but, once pain has settled, mobility can be regained by a manipulation carried out in one or more stages and under the same considerations as for a peri-arthritis which is a result of trauma.

(2) Technique of manipulation

The patient lies with the affected shoulder projecting over the side of the table. The operator, standing at the affected side, grasps the arm with one hand and places his other hand over the acromion process. An assistant fixes the axillary border of the scapula with both his hands, his arms being placed across the patient's chest. The first movement consists in powerful traction in the long axis of the arm (Fig. 5 (a)). Next, the head of the humerus may be moved forward and backward across the glenoid cavity. The following movements are then performed.

(i) *Traction with lateral rotation* (Fig. 6 (b)).—This precedes abduction because lateral rotation accompanies the abduction in normal shoulder movement; it is quite frequently found that, once full lateral rotation is

*Risk of
producing
reaction*

*Obstructed
movement*

*"Frozen
shoulder"*

restored, abduction becomes complete. The surgeon's hand exerting traction on the humerus gives the necessary steady twist.

(ii) *Traction with medial rotation.*—This is performed in a comparable manner.

(iii) *Traction with abduction* (Fig. 6 (c)).—Traction being maintained, the arm is gradually abducted. If the sudden tearing of adhesions is heard and felt, abduction is usually not continued beyond that point. If, on the other



(a) Distraction.



(b) Distraction with lateral rotation.



(c) Distraction with abduction.



(d) Adduction

FIG. 6.—Manipulation of the shoulder

hand, it is desired to ensure a full abduction range at one sitting, the final few degrees of movement are secured and confirmed by placing both the patient's arms above his head, each of his hands being held by the corresponding hand of the surgeon, who then exerts traction upward, the assistant holding the feet of the patient to prevent him from being pulled off the table.

Extension and flexion are followed by adduction of the flexed arm across the chest (Fig. 6 (d)). The movement is accomplished by grasping the lower third of the arm posteriorly, the scapula being fixed with the surgeon's other hand; the arm is pushed across towards the opposite axilla.

7. THE ELBOW

As a general rule, manipulation of the elbow with the object of regaining movement is to be avoided.

(1) Indication

Tennis elbow

This characteristic syndrome is well known. In a proportion of cases the following test is positive: the wrist and fingers are held flexed by the surgeon,

*Test of
probable
usefulness of
manipulation*

the elbow being in a semi-flexed position and the forearm pronated; forcible passive extension of the elbow, with forward pressure by the surgeon on the posterior aspect of the head of the radius, produces the familiar pain. When a positive response is obtained from this test manipulation is likely to be successful. Even if the test proves negative, however, it is worth while trying the effect of a manipulation, although the result is usually disappointing.

(2) Technique of manipulation for tennis elbow

Many procedures have been advocated for the treatment of the condition, several of which are combined in the following method. The patient's arm is grasped just below the elbow with one hand, and in the middle third of the forearm with the other. Flexion, extension, pronation, supination, abduction and adduction are then carried out rapidly and forcibly. The elbow is bent to a right angle and the wrist is forcibly flexed on the forearm in the three suc-



FIG. 7.—The classical manipulation for tennis elbow.

cessive positions of full pronation, mid-pronation and full supination. The elbow is then extended, and flexion of the wrist is repeated, the forearm being in the same three positions; the fingers are held in the position of full flexion throughout the manipulation. The classical manoeuvre is as follows. With the patient's elbow semi-flexed and with his forearm pronated, the dorsum of his hand and fingers is grasped by one hand of the operator so that the wrist and fingers are

held fully flexed (Fig. 7); the surgeon's other hand is placed on the back of the elbow, with the thumb behind the head of the radius. The surgeon suddenly hyperextends the elbow joint, forward pressure being simultaneously applied to the head of the radius with the thumb.

8. THE WRIST AND HAND

(1) Indications

(a) Chronic strain of the dorsal ligament of the wrist

Chronic pain occurring in the wrist for some time after injury or occupational over-use is a common complaint. Such conditions as teno-synovitis, undiagnosed fractures of the carpal scaphoid, or osteochondritis of the lunate (Kienbock's disease), are amongst the common causes of this symptom. Once these conditions have been excluded there remains a group of cases in which chronic strain of the carpal ligaments may be diagnosed. These cases respond well to manipulation.

(b) Chronic strain of the hand

When the hand is outstretched the palm is flat and in gripping it must become concave from side to side. Injuries or infections of any of the joints of the hands or fingers may cause secondary stiffening of other joints from reactionary oedema or disuse, so that loss of function may be more than the local lesion would produce alone.

*Causes of
pain*

9. TEMPORO-MANDIBULAR JOINT

(1) Indications

(a) *Clicking jaw*

This familiar condition may in time produce chronic strains of the capsular ligaments of the temporo-mandibular joint. These strains are painful and can be relieved by manipulation. More frequently the patient complains, not of real pain, but of the unpleasant noise and discomfort. Manipulation does not eliminate the click although it may have a good psychological effect.

*Psychological
effect*

(b) *Locked jaw*

One or other of the temporo-mandibular joints may become locked from swelling or displacement of the intra-articular fibrocartilage. It is often a matter of some difficulty to decide which joint is at fault. Manipulative reduction of the derangement should be attempted but if it fails operation for removal of the fibrocartilage is indicated.

(2) Technique of manipulation

A gag is inserted at either angle of the jaw. The handles, directed backward, are steadied by the assistant. The surgeon grips each side of the lower jaw between the finger and thumb of each hand; the thumbs, protected by gauze swabs, are applied to the teeth, and the fingers lie curled up beneath the lower margin of the jaw. The symphysis menti and body of the mandible may now be readily moved in all directions; the following movements are imparted to the temporo-mandibular joints, the gags acting as fulcrums.

(i) Distraction, by elevation of the chin. (ii) Forward and backward gliding of both condyles simultaneously, by moving the chin forwards and backwards. (iii) Forward gliding of one condyle simultaneously with backward gliding of the other, and vice versa, by rocking the chin from side to side. (iv) Lateral gliding, by moving the mandible bodily from side to side.

My thanks are due to Messrs Eyre & Spottiswoode for permission to use the illustrations redrawn from those in *Treatment by Manipulation* by H. Jackson Burrows and W. D. Coltart

[References to other titles are given under Manipulative Surgery, in the Index Volume]

MEDIASTINUM

BY N. R. BARRETT, M.CHIR., F.R.C.S.

**SURGEON TO OUT-PATIENTS, ST. THOMAS'S HOSPITAL; ASSISTANT SURGEON,
HOSPITAL FOR DISEASES OF THE CHEST, BROMPTON, LONDON**

[illegible]

1. THE SURGERY OF THE MEDIASTINUM

230.] The mediastinum has been the last anatomical region to come within the ambit of surgical treatment. This advance has been achieved at a time when the incidence of tumours affecting the region is on the increase and when surgical treatment offers good hopes of cure. Blades (1946) reported on a series of 109 patients, suffering from mediastinal tumours, upon whom he operated in a period of 3 years. Many of these tumours were diagnosed as a result of routine radiography in the United States Army; 94 were benign, 15 were malignant and, of the former, 89 tumours were successfully removed. In the past, progress has been held back because the mediastinum was a no-man's-land containing viscera, such as the heart, which had always seemed to be beyond surgical endeavour, because it was guarded from the front and the back by barriers of bone and from the sides by the pleural cavities which were unfriendly territory for the surgeon, and because it was the subject of certain physiological bogies—such as “mediastinal flutter” and “mediastinal shock”—which, however troublesome in work on animals, cause no anxiety to the surgeon who operates upon man. The question of access was, in effect, the main difficulty and this hinged upon the anaesthetic problem, the principles of which have now been solved. The surgery of the mediastinum includes the treatment of congenital abnormalities of the heart and the great vessels, of myasthenia gravis and of many pathological conditions of the oesophagus—subjects which are considered elsewhere in BRITISH SURGICAL PRACTICE.

2. MEDIASTINOTOMY

(1) Anaesthesia

Deliberate and safe operations in the mediastinum depend upon expert anaesthesia, and although the fundamental principles of surgical technique are the same in this region as in others, the difficulties which may confront the anaesthetist are greater. If, during the course of the operation, neither pleural cavity will be opened the patient may be given a spinal or a local anaesthetic. If one pleural cavity is certain to be laid open, as for example in the case of a patient who elects to operate without an anaesthetic, the other pleural cavity will be open at the same time—as when the pulmonary ligaments on both sides are dissected out for the removal of a carcinoma at the lower end of the oesophagus—the situation can be safely controlled only by the anaesthetist who has the patient under general anaesthesia and connected with a circuit which allows controlled or artificial respiration.

(2) Exploration of the superior mediastinum

cervical
ach

the sternum, through the usual cervical approach. If the goitre is large, so that it cannot be shelled out with the finger, a spoon or the aspiration of a cyst often solves the problem. “Blind” operations of this type are safe because the adenoma is separated from the large mediastinal vessels by its capsule, but to remove any other “tumour” by this means is to perform an operation which will be incomplete and dangerous.

The superior mediastinum can also be approached from an incision above the middle of the clavicle. Operations for the removal of pharyngeal pouches, and other conditions, have shown that the pleura, which extends into the neck as high as the transverse process of the first thoracic vertebra, can easily be stripped outwards from the sides of the bodies of the vertebrae by gauze dissection, thus exposing the side of the superior mediastinum as far down as the fourth thoracic vertebral body. This manoeuvre can be useful during operations upon the upper three thoracic sympathetic ganglia.

A limited exploration of the superior mediastinum can be done from the back, which is the method of some who operate upon cervical ribs or conditions such as congenital atresia of the oesophagus. The approach is extra-^{Extrapleural approach} pleural and is performed as follows. The patient is laid in the lateral position with the arm held well forward so that the space between the vertebral border of the scapula and the midline is as large as possible. A vertical incision—straight or "J"-shaped—about 6 inches long is made between the midline posteriorly and the back of the scapula. The trapezius and short rotator muscles are incised in the line of the incision, exposing the back ends of the ribs. A short length of one rib, the latter varying according to whether access is required to the upper or the lower parts of the superior mediastinum, is removed subperiosteally, care being taken not to open the pleura. The periosteal bed of the rib is incised and the outer surface of the parietal pleura exposed; the latter can then be stripped off the chest wall either directly forwards if a limited exposure is needed, or from the whole of the upper part of the thoracic cage, thus exposing the structures in the mediastinum from the dome of the pleura to the hilum on the right and to below the aortic arch on the left. When the operation in the mediastinum has been finished, the chest wall is closed in such a way as to be air-tight and air or liquid remaining in the operation space is aspirated. A similar but wider access to the side of the superior mediastinum is obtained by upper thoracoplasty when this operation is combined with an extrapleural or an extrafascial stripping.

(3) Exploration of the posterior mediastinum

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exposure is necessary. The patient lies on one side and the pleural cavity is

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gives a limited exposure, and it is practically confined to the drainage of
collections of pus in the posterior mediastinum. After the abscess—^{tuberculous}
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border

Laparotomy

exposed and the head and neck of the rib together with the corresponding transverse process are removed. To do this the erector spinae must be separated from the outer aspect of the rib and of the transverse process with a rongeur and the muscle retracted away from the bones. The portion of rib is excised with rib-cutting forceps, and bone removal is completed with nibblers. The mediastinum is reached by blunt dissection, keeping close to the bodies of the vertebrae and displacing the parietal pleura inwards. A limited exposure of the lower posterior mediastinum can be made by laparotomy. The lower 2 inches of the oesophagus can be exposed by mobilizing the viscus in the diaphragmatic pinchcock and drawing it downwards by traction. This approach is sometimes used to perform Heller's operation for achalasia and vagotomy for peptic or anastomotic ulcer.

(4) Exploration of the anterior mediastinum*(a) Anterior transpleural thoracotomy*

The patient lies on his back and an incision parallel to the sternum and about 1 inch from its edge is made from the second rib anteriorly to the fourth rib, at which point the incision turns outwards along the fourth intercostal space. The pleural cavity is opened by incising the costo-chondral junctions of ribs two and three, and carrying the incision outwards along the intercostal space. The internal mammary vessels are ligated and divided if necessary and the flap of anterior chest wall thus created is lifted upwards and outwards. The pleural cavity is opened and an excellent exposure of the side of the upper mediastinum is obtained. At the conclusion of the operation the chest is closed by fixing the costo-chondral junctions back in place with thread stitches and by sewing the intercostal muscles.

Alternative anterior approach

An alternative anterior approach is that used by Blalock in the treatment of pulmonary stenosis, and by some surgeons for ligation or division of a patent ductus arteriosus. The pleural cavity is opened by a horizontal incision passing outwards from the sternal edge along the second intercostal space to the axilla. The ribs are spread and the mediastinum is exposed across the pleural cavity after the upper lobe of the lung has been retracted. This incision gives good access for operations upon the blood-vessels in one side of the mediastinum but has the disadvantage that the scar is likely to become keloid, and in women it cuts through the upper part of the breast. To avoid the latter a lower incision may be used in the skin and a flap of pectoral muscles and breast dissected upwards as far as the second intercostal space.

... .. the sternum

... .. of the operation has been performed. There are occasions, such as the removal of the thymus for myasthenia gravis, or the removal of some anterior mediastinal tumours, upon which a general exposure is necessary, and for these it is wise to split the sternum. This operation is more serious than others because it produces, post-operatively, a bilateral hindrance to respiration; but it causes little shock, it is easy to perform and the divided sternum heals well. The patient is given a general anaesthetic and an intratracheal tube with a cuff is passed so that artificial respiration can be maintained if one or both pleural cavities are opened. A small collar incision is made low in the neck and from

the middle of this a vertical limb is carried down exactly the centre of the sternum. The bone is split either with special shears, which resemble those used to remove plaster of Paris, or a chisel, and the split is carried from the jugular notch to the level of the third or fourth costal cartilage. It is unwise to split the sternum from the jugular notch to the xiphoid process because if an intact hoop of ribs, vertebrae and costal cartilage remains at one place this facilitates efficient respiration during convalescence. The two halves of the sternum should be separated gradually with a rib-spreader and whilst this is being done care should be taken not to injure or tear the pleural membranes, both of which lie near the midline anteriorly; they can be displaced either by sharp or blunt dissection. There is very little bleeding and an excellent view of the great vessels, the thymus and the front of the pericardium is obtained. At the conclusion of the operation the two halves of the sternum can be held in apposition by circumferential stitches or by passing one stout stitch through the cartilaginous junction between the manubrium and the body of the bone. Pain during convalescence can be relieved by injecting a small amount of Proctocaine into the front end of each of the upper intercostal spaces and by firmly strapping the front of the chest to prevent movement at the fracture.

3. ENLARGED MEDIASTINAL GLANDS

(1) Anatomy and effects of enlargement

The lymph glands in the mediastinum become abnormally enlarged as a result of pathological conditions affecting the lungs, the oesophagus and the lymphatics of the body in general. The glands concerned are situated in the superior, the posterior, and the anterior mediastinum and connect with the lower deep cervical, the supraclavicular, the glands in the lesser omentum and those in the hilum of the liver. The bronchial glands are disposed in three main groups and are concerned with drainage of the lungs. The right tracheo-bronchial group lies in the superior mediastinum to the right of the trachea and above the azygos vein, enlargement displaces the trachea; in malignant disease they may cause paralysis of the phrenic nerve but not tracheal obstruction. By contrast, enlargement of the left tracheo-bronchial group is apt to cause obstruction to the left main bronchus because the glands lie to the left of the trachea, around the left main bronchus, and are confined by the arch of the aorta above and the pericardium below; the left phrenic and the left recurrent laryngeal nerves pass through this space. The inferior tracheo-bronchial group lies in the angle between the diverging right and left stem bronchi, enlargement causes distortion and widening of the carina, but seldom leads to complete obstruction of either stem bronchus. In addition to the main groups there are smaller units situated in the angle caused by every bifurcation of a main bronchus in the hilum of the lung. A few other glands are found in the anterior mediastinum in relation to the internal mammary vessels, but these seldom concern the surgeon except when a carcinoma of the upper and inner quadrants of the breast metastasizes to glands in the front of the second intercostal space (Handley, 1947) or when, becoming infected with tuberculosis, a collar-stud abscess forms and tracks round in the endothoracic fascia to present as a cold abscess of the chest wall (Barrett, 1939).

FIG. 10—Mediastinaladenitis. Skiagram of the chest of an infant, showing a large mass of glands in the superior mediastinum, causing atelectasis of the middle and lower lobes of the right lung.

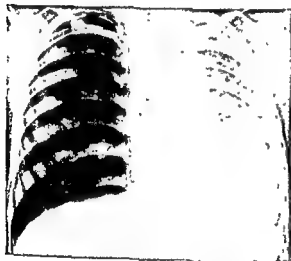


FIG. 11—Mediastinaladenitis. Complete atelectasis of the left lung of a child aged 1 years, caused by mediastinaladenitis. Cases of this type proceed to permanent pulmonary lesions, such as bronchiectasis, if not treated during the stage of atelectasis.

FIG. 12—Malignant mediastinal glands. This patient, a middle-aged man, has a large mass of malignant glands projecting from the mediastinum, particularly on the right side, and causing complete atelectasis of the lower lobe of the right lung.



disease affecting mediastinal glands; paralysis of nerves is an unusual complication of inflammatory disease in the mediastinum.

*Superior vena
caval
obstruction*

Superior vena caval obstruction is suggestive of secondary malignant disease affecting the mediastinal glands; but vena caval obstruction can be produced by conditions such as simple or malignant retrosternal goitre, spontaneous thrombosis of the superior vena cava (Tubbs, 1946), actinomycosis of the mediastinum (Mounsey, 1947) and malignant tumours of the thymus. These lesions produce abnormalities which may be confused with enlarged glands.

*Tuberculous
glands*

Tuberculous glands in the superior mediastinum are generally a part of a "primary complex", but can occur without evidence of pulmonary tuberculosis. They arise and develop in adults in the same way as do tuberculous glands



FIG. 13 — Lymphosarcoma of mediastinum. Skiagram of the chest of a woman, aged 40, with lymphosarcoma of the mediastinum. The tumour was producing signs and symptoms of pressure at the upper thoracic outlet. The mass disappeared clinically as a result of two courses of radiotherapy; the patient has remained well for 3 years.

in the neck and may form a "tumour" causing pressure symptoms in the thoracic outlet, or they may caseate and break down into a cold abscess in which the pus is often inspissated. These patients hardly ever need surgical treatment but if a mediastinotomy is performed in error the glands should not be removed, because they are closely adherent to the innominate veins.

Sarcoidosis is nearly always associated with lesions in the lungs, the skin and elsewhere in the body, and pneumoconiosis is caused by certain occupations and is demonstrated by radiography.

Barnard (1926) showed that primary lymphosarcoma of the mediastinum (Fig. 13) is a rare disease, and that in most cases the mediastinal deposits are metastases derived from oat-celled carcinoma of the bronchus. Lymphadenoma and other lymphomatous tumours occur, however, and, although other manifestations of these diseases may be present, mediastinal glands can be the only demonstrable abnormality. In this group of diseases a course of radiotherapy is valuable as a diagnostic measure, because relief of symptoms generally occurs soon after treatment has been started and serial skiagrams show a rapid reduction in the size of the mediastinal opacities. *Mediastinal deposits*

(4) Treatment

In infants many cases are "primary" and due to non-specific organisms. They generally resolve quickly so that surgical treatment is not required; others are a part of a primary tuberculous complex. Whatever the cause may be, bronchial obstruction is often the result and if atelectasis of a lung or a lobe of the lung develops and persists, bronchoscopy should be performed. The lumen of the obstructed bronchus can often be re-established by painting oedematous mucosa or granulation tissue with a weak solution of cocaine and adrenaline, and the air passages beyond the obstruction can be cleared by sucking out pus and mucus, or by aspirating the contents of a tuberculous gland which has ulcerated into the bronchus. Efficient, and perhaps repeated, treatment on these lines can avert the subsequent development of bronchiectasis but it is necessary to stress that bronchoscopy in infants can be a dangerous operation unless it is performed with the utmost gentleness and skill. *Bronchial obstruction*

When symptoms of obstruction are due to lymphadenoma or lymphosarcoma, a marked temporary relief can be obtained by radiotherapy and the same is true of superior vena caval obstruction caused by metastases in the superior mediastinum from carcinoma elsewhere in the body (for example, in the breast or the thyroid gland).

Carcinoma of the bronchus or of the oesophagus, complicated by secondary deposits in glands, is generally inoperable and only likely to be temporarily benefited by radiotherapy. Operations upon both these conditions have shown, however, that such glands can often be safely removed in one block with the tumour and that all the enlarged glands are not necessarily carcinomatous. *Carcinoma*

4. MEDIASTINAL EMPHYSEMA

(1) Aetiology

Surgical emphysema in the mediastinum is a rare but important complication of many different pathological conditions. The air, which comes from the tracheo-bronchial system, the oesophagus, or the surface of the body, is sucked or pumped into the mediastinum by the movements of respiration and spreads easily in the areolar tissue between the viscera and blood-vessels; it seldom passes below the diaphragm, being held at the crura, but travels upwards to reach the subcutaneous tissues at the base of the neck. The causes of mediastinal emphysema are spontaneous, pathological, or traumatic rupture of the oesophagus at any level; wounds of the lung or of the air passages and sometimes operations such as tracheotomy or the making of bronchograms by the crico-thyroid membrane technique; the sudden introduction *Rupture of oesophagus*

into the lungs of anaesthetic gases under pressure; or violent coughing such as occurs in children suffering from whooping-cough or asthma (Fig. 14).



FIG. 14.—Skiagram of the chest of a child showing extensive subcutaneous and mediastinal emphysema. The patient was suffering from broncho-pneumonia (*Dr. A. G. Watkins's case.*)

(2) Diagnosis

The diagnosis is important partly as a pointer to the underlying cause and partly because treatment may be necessary as an emergency. The air bubbles in the subcutaneous tissues can be palpated at the base of the neck or the typical crackling sound may be heard on auscultation. The diagnosis can often be made from skiagrams in which the air can be seen in the mediastinum or lying between the heart and the lungs as a rim around the pericardium.

(3) Treatment

Treatment is of the lesion which caused the emphysema, but sometimes this lesion develops so quickly and is so extensive that death from suffocation and from pressure upon the great veins—which prevents the heart from filling—can occur unless prompt action is taken by the surgeon. Most of the cases leading to acute mediastinal obstruction have been reported after sudden

pressure anaesthesia; when this state exists it does not suffice to remove the anaesthetic apparatus, because the hole in the air passages has been produced and the increasingly violent attempts at respiration on the part of the patient augment the emphysema so that it may continue to spread and to asphyxiate him. Relief can be given by incising the deep fascia in the base of the neck above the jugular notch; as an alternative a wide-bore aspirating needle can be introduced beyond the deep fascia in the supraclavicular area and suction applied (Barrett and Thomas, 1944). *Incision of deep fascia*

5. THE THORACIC DUCT

The thoracic duct carries the chyle and the greater part of the lymph from the abdomen and the lower extremities to the left subclavian vein. Theoretically occlusion of, or injury to, this duct should have serious consequences, but numerous alternative pathways exist, and if the main channel becomes involved in a fistulous track it can safely be divided or ligated in any part of its course (Olsen and Wilson, 1944). Pathological conditions involving the thoracic duct are manifest when a chylothorax or a superficial fistula has developed. One-third of all cases of chylothorax are due to trauma; the condition can also be produced by severe coughing, by crush injuries involving the thorax, by hyperextension injuries of the spine and by any operation performed in the territory traversed by the duct. Chylothorax of non-traumatic origin is generally due to malignant disease involving the duct, the innominate and the subclavian veins, or the mediastinal lymph glands. It can also be shut off by tuberculous or carcinomatous deposits secondary to tuberculosis or cancer of the abdominal viscera. The surgery of the thoracic duct is chiefly concerned with the management of chylous fistulae and these are prone to occur at three different levels. *Chylothorax*

Chylous fistulae

(a) In the neck

Operations to paralyse or avulse the phrenic nerve, or block dissections at the base of the neck can be complicated by an injury to the thoracic duct. This is seldom noticed at the time but becomes obvious a few hours later when the wound swells up and begins to discharge chyle. Surgical treatment is not indicated because the fistula closes spontaneously within a few days.

(b) In the upper mediastinum

A fistula in the upper mediastinum is uncommon but is a complication of left upper thoracoplasty with apicolysis. The thoracic duct is not generally seen during an extrafascial stripping of the apex of the lung but sometimes a small collection of milky liquid appears in the bottom of the wound, and when this has been mopped away a tiny hole in the side of the duct is apparent; in such a case the duct should be tied above and below the perforation. More usually the injury passes unnoticed at the time and is revealed either because a large collection of liquid, which looks like white pus, accumulates in the wound and is aspirated or is found by chance at the second-stage thoracoplasty. The presence of such a collection causes few constitutional signs except pyrexia and is no contra-indication to proceeding with the thoracoplasty. The important point is that the chylous exudate should not be mistaken for pus, for to drain such a collection does much harm. Chyle looks *Importance of differentiating from pus*

whiter than pus; it begins to collect within a few hours of the operation; the patient does not look as though a large acute abscess is present in the operation wound; and if the liquid is mixed with ether it becomes practically clear. If doubt exists in the diagnosis between chyle and pus a few drops of the liquid can be stained with Sudan III.

(c) *In the posterior mediastinum*

Aspiration

Exploration of mediastinum

A fistula in the posterior mediastinum does not lead to diagnostic symptoms until the chyle has burst through into one or both pleural cavities. The common cause of such an accident is an operation performed in the vicinity, such as the removal of a large chondroma of rib or vertebra. In such cases the chyle accumulates quickly in the pleural cavity and its volume is augmented by a pleural effusion which is poured out as a result of irritation. Aspirations relieve the pressure symptoms but the patient, losing fat and proteins rapidly, goes downhill and conservative measures are of no avail. Attempts have been made to meet this situation by repeated aspirations and by reintroducing the liquid into the patient's veins, but this practice is dangerous and fatal reactions have been recorded. The correct treatment is to explore the mediastinum. Under general anaesthesia the pleural cavity should be opened by a wide lateral thoracotomy, and the liquid in the pleura should be aspirated, after which the track in the mediastinum can generally be seen and the thoracic duct can be ligated. It is said that if the fistula cannot be seen the chyle issuing from it can be dyed red by introducing some fat and Sudan III into the small intestine through a stomach tube and waiting a few moments.

6. ACUTE SUPPURATIVE MEDIASTITIS

Suppurative mediastinitis is a rare condition, which until recently was regarded as fatal. In fact the majority of cases can be saved by prompt surgical intervention.

(1) Aetiology

The most complete treatise on this subject is that by Neuhoff and Jeremyn (1943) who classify the causes as follows:

Perforation of oesophagus

Forty per cent of all cases are due to perforation of the oesophagus by endoscopy, impaction of a foreign body, gastroscopy or wounds. Twenty-eight per cent are due to suppuration spreading from acute inflammations in the pharynx, the cervical vertebrae, cervical glands and suppurative thyroiditis. The remainder are due to such conditions as spontaneous perforation of the normal oesophagus or perforation of the oesophagus affected by carcinoma, peptic ulceration, oesophagitis and other conditions. Very few are the result of suppurative lymphadenitis secondary to inflammations in the lungs, the pleural cavities or the pericardium.

(2) Anatomy

The space in which these inflammations occur extends from the pharynx to the diaphragm and the tissue concerned is the loose areolar tissue which surrounds the oesophagus and the sheaths of the great vessels. This space presents no obstruction to spread of liquid or gas under tension; but sup-

astinum. The space is bounded laterally throughout the whole of its extent

extent by the two pleural cavities and contains the great vessels and the heart, the constant motion of which not only favours the spread of pus but is detrimental to inflamed tissues.

(3) Pathology

As perforation of the oesophagus is the commonest cause of suppurative mediastinitis, this aspect of the subject will be considered. The effect of perforation depends primarily upon the speed at which it occurs and whether or not the accident is secondary to disease in the gullet. If slow, adhesions form and the process may be limited to an abscess in the mediastinum; this may remain relatively quiescent or may resolve spontaneously but the abscess is more likely to increase and to rupture into the pleural cavity or into an adjacent viscus. If rapid perforation occurs as, for example, from a penetrating wound or endoscopy, cellulitis develops and not only spreads through the mediastinum but, as tension rises, the inflammatory exudate may rupture into one or both pleural cavities; death supervenes within a few hours. The life of the patient depends upon prompt diagnosis and treatment.

Prompt diagnosis and treatment necessary

(4) Diagnosis

Two types of mediastinitis will be considered. First, there is perforation due to operation or trauma. The commonest cause is an attempt to pass an oesophagoscope through the crico-pharyngeal sphincter when the latter is in spasm; the rupture is in the neck and, if untreated, the inflammation rapidly spreads to the superior mediastinum. Diagnosis is suggested by the following points (Phillips, 1938). There is a history of injury or of difficult instrumentation. Pain, tenderness, dysphagia, fever, possibly a swelling in the neck and surgical emphysema are early manifestations. A barium swallow, or preferably examination after swallowing a little Lipiodol, often shows the level of the perforation. Endoscopy may be necessary in doubtful cases.

Perforation due to trauma

The second type is that which complicates spontaneous perforation of the lower oesophagus (Barrett, 1946). This catastrophe generally occurs in drunken men during a violent fit of vomiting but the sequence of events which results is typical of other perforations in the posterior mediastinum. The patient experiences an excruciating pain which has been described as though the heart was bursting, and this is followed by collapse and symptoms which closely resemble those of a perforated gastric or duodenal ulcer. When the liquid in the mediastinum ruptures into the pleural cavity an effusion rapidly develops; cyanosis, dyspnoea and surgical emphysema, which rises up into the base of the neck, are confirmatory signs. Skiagrams taken soon after the onset of symptoms may show no abnormality but a pyopneumothorax is soon apparent and surgical emphysema may be demonstrated. Aspiration of acid liquid containing particles of food from the mediastinum or the pleural cavity is diagnostic. The expectation of life without surgical treatment is about 24-48 hours during which time the patient is in continuous agony and frequently is erroneously treated as having a perforated gastric ulcer.

Complication of spontaneous perforation of lower oesophagus

(5) Treatment

All perforations of the oesophagus, except those seen late, which have localized spontaneously, require immediate surgical treatment; such treatment practically assures recovery. The principles are to localize the site of the

perforation and, having exposed it, to close the hole in the gullet, to do a gastrostomy by which to feed the patient while the perforation heals, to lay open and drain the infected tissues widely and to administer sulpha drugs and antibiotics.

Mediastinitis originating in the neck should be operated upon by exposing the oesophagus through a long incision parallel to the anterior border of the sternomastoid; the thyroid gland is displaced forwards and the peri-oesophageal tissues are laid open. The perforation in the gullet is closed with inverting stitches of wire, thread or catgut and the infected tissues are drained with a piece of rubber drain and treated with sulpha drugs and penicillin. The wound is left open until the danger has passed, when secondary suture may be practised. If, during the operation, the infection is found to have burrowed down into the superior mediastinum, counter-drainage can be established by a costo-transversectomy at the level of the third rib on the side which is chiefly affected. It is important not to injure the pleural membrane during this operation.

Counter-drainage by costo-transversectomy

If the chief site of the cellulitis is the posterior mediastinum the best approach is by wide lateral thoracotomy. In this way the whole of the mediastinum can be exposed, tension can be relieved by incising the mediastinal pleura from top to bottom and by draining the infected material into the pleural cavity. Penicillin and sulpha drugs are dusted in the mediastinum. The thoracotomy wound is sewn tightly in layers, but closed drainage of the pleura is provided by a water-seal as in the treatment of early total pleuritis.

Localized abscess

A localized abscess anywhere in the posterior mediastinum is best treated by costo-transversectomy and a similar abscess in the anterior mediastinum can sometimes be appropriately drained by removing the inner end of the clavicle.

7. PRIMARY TUMOURS OF THE MEDIASTINUM

Under this heading will be considered a number of different tumours which develop in the mediastinum and cause symptoms by exerting pressure upon adjacent structures.

(1) Dermoid cysts and teratomas

The commonest tumours in this group are dermoid cysts and teratomas. Reviewing the literature up till 1939, Rusby (1944) found that 245 cases had been reported; large numbers of other cases have been treated by surgeons who have not recorded their experiences. The term "teratoid" has been suggested by Harrington (1929) because these tumours contain cells representative of the three embryological germ layers; it is, however, convenient to speak of "dermoids" which are predominantly cystic and simple and "teratomas" which are largely solid and prone to malignancy; this division is not rigid. The majority of the tumours grow in the anterior mediastinum and, by encroaching upon the superior mediastinum, cause pressure symptoms affecting the trachea and bronchi, the great veins, the nerves and even the oesophagus. The severity of these symptoms varies with the simple or malignant nature of the tumour, and its position rather than with its size.

Pressure symptoms

(a) Pathology

(i) *Dermoid cysts*.—These are usually simple and consist of a single cavity which may be lobulated. The cyst contains sebaceous material and is lined

with squamous or columnar epithelium which rests upon other tissues typical of normal dermis and epidermis. In one or more parts the wall of the cyst is solid and it is in these areas that mesodermal and endodermal elements are often found. Hair follicles are much in evidence and strands of hair—which are often of different colour and texture from that of the patient's hair—float in the sebaceous material. On opening the cyst a peculiar odour emanates from it. The cyst nearly always has a pedicle, the size and vascularity of which varies, but which is attached to the mediastinum in the neighbourhood of the left innominate vein. The wall of the cyst varies in thickness according to its size and age; it may contain plaques of calcification or bone and is lightly or densely adherent to adjacent structures, such as the pericardium or the pleura. *Adherence to adjacent structures*

(ii) *Teratomas*.—These vary enormously in appearance and in malignancy. At one end of the scale are tumours which look like dermoid cysts but which, on microscopy, prove to be malignant; these, if left untreated, alter their character and become more solid as the malignant elements multiply. At the other pole is the complex teratoma which has been described as a parasitic foetus. Almost all retain some cystic areas and these are filled with mucus, blood or degenerating tumour cells. The surface of the tumours is lobulated and often hard; the surrounding tissues are oedematous, and the adjacent structures are invaded so that surgical removal becomes impossible.

Both dermoid cysts and teratomas can grow to an enormous size before any outward deformity or symptoms attract the attention of the patient. Their pathogenesis is not known, but it is generally assumed that they are present at birth and, remaining dormant at first, begin to grow very slowly at puberty and come to light between the ages of 20 and 40.

(b) *Diagnosis*

Many of these tumours are being discovered by mass radiography before symptoms have occurred, but progressive enlargement gradually produces pressure symptoms of which the usual are cough, dyspnoea, pain, engorgement of the veins in the neck, palpitations of the heart, paralysis of the diaphragm, Horner's syndrome and changes in the voice. To these may be added *Symptoms*

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patient coughs up hairs, sebaceous material, pus and debris. Infection can occur without a bronchial communication, and a bronchial communication is not certainly followed by infection. Upon occasion the first sign is the appearance of a tumour growing from behind the sternum into the base of the neck and sometimes the onset of malignancy produces signs and symptoms which mark the first change from normal health. The physical signs are those of a mass in the anterior or superior mediastinum, and there may be evidence of pressure upon, or invasion of, adjacent structures. Examination of the sputum may reveal hairs or sebaceous material. In doubtful cases the clinician may be tempted to aspirate the mass but this seldom gives information and is dangerous. Radiography is of great importance and films taken in two planes must be secured. The tumours are generally globular and clear-cut but, on the whole, dermoid cysts are spherical whereas teratomas are bossed. *Onset of malignancy*

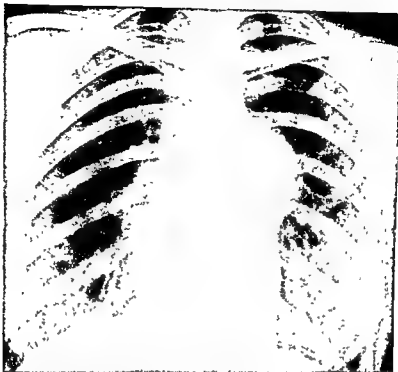


FIG. 15.—Mediastinal dermoid. Skiagram of the chest of a woman showing pulmonary tuberculosis in the left lung and a mediastinal dermoid cyst. In the latter there is a fluid level and some air. (Mr. C. Price Thomas's case.)



FIG. 16.—Mediastinal dermoid cyst. Skiagram of the chest of a young fit man who was sent to the hospital as a case of aneurysm of the aortic because the tumour, which can be seen protruding from the mediastinum in the vicinity of the right side of the heart, was found to pulsate on screening. A simple dermoid cyst was removed by anterior mediastinotomy.

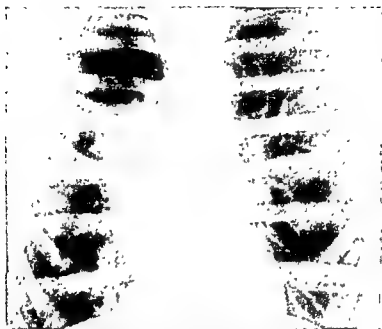


FIG. 17.—Teratoma of the mediastinum. Skiagram of the chest of a middle-aged man showing an irregular and long opacity projecting into the right-lung field from the mediastinum. Two teeth can be seen at the top of the opacity.



FIG. 18.—Teratoma of the mediastinum. Lateral skiagram of the same patient as in Fig. 17 showing that the tumour lies in the anterior mediastinum and contains several fully formed teeth.

The wall may show calcification or bone and the mass may contain opaque elements such as teeth. If rupture has taken place, a fluid level above which there is air will be found (Fig. 15), and fluoroscopy will reveal pulsations if the cyst lies near the heart—a common fault in diagnosis is to mistake a dermoid cyst for an aneurysm of the auricle (Fig. 16). Rusby (1944) states that a positive Aschheim-Zondek test in a man with a normal testis, or in a female if pregnancy can be excluded, together with the radiological demonstration of an anterior mediastinal opacity (Figs. 17 and 18) would go a long way towards disclosing the teratomatous, and probably malignant, nature of a tumour containing chorion-epitheliomatous elements.

(c) Treatment

The treatment of all anterior mediastinal tumours is surgical removal as soon as diagnosis has been made; to this rule there are no exceptions unless the patient is moribund. Neither size of tumour nor severity and type of pressure symptoms are contra-indications. The results of surgical treatment are good; the result of delay is generally death.

Operative methods

Dermoid cysts are apt to present to one side of the mediastinum and can practically always be taken out through an anterior transpleural thoracotomy or by opening the anterior mediastinum, having divided one or more costal cartilages and displaced the intact pleura outwards. If the cyst is large, a lateral thoracotomy may be preferred as affording better access.

Teratomas may be dealt with in the same way but a sternal split may be better—particularly if the tumour lies in the midline, or is bilateral and of doubtful type.

Infection

Infection in a dermoid cyst should be controlled before any attempt is made to remove the tumour. This can be done by external drainage if postural methods and antibiotics fail to secure adequate bronchial drainage. If prolonged sepsis has caused pulmonary suppuration it may be necessary to do a lobectomy or a pneumonectomy as well as to remove the tumour.

In all these tumours the pedicle lies in close relation to the left innominate vein but multiple adhesions, either inflammatory or malignant, to vital structures may make extirpation impossible. In such cases partial removal of the tumour can afford temporary relief but the results are bad.

Aspiration, drainage and radiotherapy alone, as methods of treatment, are contra-indicated.

(2) Mediastinal lipoma

These tumours originate in the pre-pericardial pad of fat in the angle between the sternum, the pericardium, and the diaphragm, or from the fat in the superior mediastinum. Two different varieties have been described, namely, those which are entirely intrathoracic and those which have processes extending outwards between the ribs. Both give rise to lobulated tumours which remain encapsulated but which ultimately may become sarcomatous. A large lipoma may cause a great deal of pressure upon the heart and great vessels, but it does not cause symptoms until late, so that the mere size of the tumour is a point in favour of the diagnosis. Pressure symptoms are not a prominent feature unless, or until, malignant change has occurred. Skiagrams may be very similar to those

found in dermoid cysts or teratomas but, on the whole, fat is less radio-opaque than is either liquid or solid cellular tissue, and the irregular outline is a point against the diagnosis of a dermoid cyst. Fibroma and fibro-lipoma also occur in the anterior mediastinum and cannot be differentiated clinically



FIG. 19—Mediastinal lipoma. Skiagram of the chest of a boy aged 4 years, with a large space-occupying mediastinal tumour which has displaced the left lung to the left and the heart to the right. A lipoma was successfully removed 4 years ago; the patient has progressed normally since the operation.





FIG. 21.—Springwater cyst of the mediastinum. Lateral skiagram of a man showing a springwater cyst situated in the angle between the pericardium, the sternum, and the diaphragm



FIG. 22.—Springwater cyst. Photograph of the cyst shown in Fig. 21, after removal.

from lipoma. The treatment is surgical removal and the particular hazards of the operation are those associated with the removal of any large mass of tissue from a relatively small space (Fig. 20). If the tumour has become sarcomatous, a combination of wide surgical removal and radiotherapy is indicated.

(3) Springwater cysts of the mediastinum

Many examples of thin-walled cysts growing in the superior mediastinum or in the pre-pericardial area have been reported (Fig. 21). They contain liquid which looks like water and in consequence they are translucent at operation and are called springwater cysts (Fig. 22). On histological section the wall consists of loose, avascular, fibrous tissue and the lining is of flattened endothelial cells. The nature of these cysts is not known, and they are generally discovered upon routine or mass radiography, protruding from the mediastinum into one or both pleural cavities. The cysts are lax so that if a

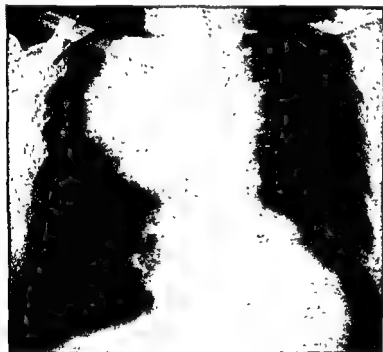


FIG 23—Paratracheal cyst. Skiagram of a man aged 58, who complained of symptoms due to thrombosis of the subclavian vein on the right side and dyspnoea. The skiagram shows a large circumscribed opacity protruding from the mediastinum. At operation this was found to be a paratracheal cyst.

diagnostic artificial pneumothorax is induced the opacity which is seen in skiagrams changes its shape and this point is of diagnostic importance. The diagnosis can often be confirmed pre-operatively, if an artificial pneumothorax has been induced, by examining the tumour with a thoracoscope. The treatment is removal, either by a lateral transpleural approach or by anterior extrapleural mediastinotomy.

Cystic lymphangiomata of the mediastinum are rare but have been described (Sanes, MacManus and Scratchard, 1945); they resemble similar lesions in the neck or the axilla and require the same treatment.

(4) Paratracheal cysts

When first discovered these cysts vary in size from a walnut to a large orange. They lie adjacent to the trachea and displace it to one side (Fig. 24). The aetiology is unknown but they are formed from tracheal elements, and all stages between a tracheal diverticulum and a separate cyst have been described. The wall is of fibrous tissue and may contain small fragments of cartilage and mucous gland, and the lining is either columnar epithelium or flattened endothelium, or is absent. The content of the cyst is mucus but, if inflammation has occurred, it may contain sterile or infected pus, and in the latter event adhesions form in the pleural cavity over the swelling. The symptoms



FIG. 24.—Paratracheal cyst. Tomogram of the same case shown in Fig. 23. This illustrates the actual size of the tumour and the degree of tracheal displacement. The differential diagnosis in such a case is from retrosternal goitre.

are due to tracheal or mediastinal pressure, and the former can usually be demonstrated in skiagrams or on bronchoscopy. The typical radiographic appearance is of a homogeneous, circumscribed opacity, based on the superior mediastinum and projecting into the pleural cavity (Figs. 23 and 24). If there is a large cyst, it may compress the pulmonary artery and cause an arterial aneurysm. Treatment is by anterior thoracotomy.

*Typical
radiographic
appearance*

(5) Mediastinal cysts of bronchial origin

These resemble the paratracheal cysts but occur lower in the mediastinum and are generally found in children in whom they have caused bronchial obstruction leading to pulmonary signs and symptoms (Brown and Robbins, 1944). The cysts are shut off from the bronchial tree but some become infected without open communication. Allison (1947) states that "...most authors agree that these cysts arise in the young embryo by the separation of a diverticulum of endoderm and mesoderm from the foregut at the point at which the tracheal bud appears, or by such a separation from the growing and branching tracheal bud itself". Microscopically the walls contain all the elements found in a normal bronchus, and the contents vary from mucus to pus. The treatment is surgical removal.

*Occurrence
in children*

(6) Gastric and enteric mediastinal cysts

These cysts occur in the posterior mediastinum and may attain considerable size. The gastric cysts are lined by gastric mucous membrane and their walls may contain all the muscle elements typical of stomach. As the cyst increases in size, acid and pepsin are secreted into the lumen and a typical gastric ulcer or haemorrhage can occur. The contents may be milky, bloody or clear fluid which is acid or neutral in reaction. Enteric cysts resemble small intestine.



FIG 25—Skiagram of a man aged 51 years, who had right-sided thoracic pain, showing a solitary neurofibroma which has displaced and eroded the fourth rib posteriorly. Tumour successfully removed by lateral thoracotomy.



FIG. 26.—Von Recklinghausen's disease. Skiagram of a young girl showing an opacity at the apex of the right chest. Operation disclosed multiple neurofibromatosis involving the mediastinum and the intercostal nerves. (*Mr. Price Thomas's case.*)



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cell for

In both varieties the cyst is separate from the oesophagus and often occurs behind the hilum of the lung to which it becomes adherent. Dense adhesion to the diaphragm and erosion of the bodies of adjacent vertebrae have been reported. The diagnosis is difficult before operation, but is suggested by the skiagrams taken in the antero-posterior and lateral planes, and is fairly certain when a mesenteric cyst of similar type is found in the abdomen of a patient who has radiologically a circumscribed opacity in the posterior mediastinum. The treatment is removal by lateral thoracotomy.

*Adhesions
and erosion of
vertebrae*

(7) Mediastinal tumours of neurogenic origin

This important group of tumours includes a number which are relatively common. There is no agreed classification, but the following serves ordinary clinical purposes.

(a) *Solitary neurofibroma*

This is a benign tumour which arises from the neurilemma sheath of Schwann, or the connective tissue sheath of any intercostal nerve. It generally attains the size of a cricket ball before becoming clinically apparent; it lies in the posterior mediastinum and the paravertebral gutter, and tends to cause "spreading of the ribs" and erosion by pressure (Fig. 25). Clinically it leads to pain and, if the tumour has been present for a long time and is large, the lung may be adherent and haemoptysis can occur; in cases of this type, haemo-

Haemoptysis

(b) *Multiple neurofibromatosis*

Von Recklinghausen's disease affects the intercostal nerves both in the posterior mediastinum and in the intercostal spaces (Figs 26 and 27). The tumours can grow to an enormous size and are prone to sarcomatous change. The patients seldom complain of any symptoms until late in the disease, when a large space-occupying thoracic lesion causes respiratory embarrassment, or pain develops as a mark of malignant change. The intrathoracic tumours may be the only manifestation of multiple neurofibromatosis or may be part of a general disease. When symptomless and discovered during the course of a routine examination, it may be tempting not to advise operation but if there is reasonable prospect of removing all the intrathoracic tumours this should be done to avert the risk of sarcoma. The tumours should be explored and removed by lateral thoracotomy.

*Sarcomatous
change*

(c) *Dumb-bell tumours*

Dumb-bell tumours are those which grow in relation to the intercostal nerves as the latter emerge from the vertebral column. They are generally simple, and lie partly within and partly without the vertebral canal. By pressure they often cause erosion of the foramen concerned and may be so extensive as to involve several nerves. If the tumour grows principally within the vertebral canal, signs and symptoms of pressure upon the cord appear. The treatment is surgical removal. The thoracic part of the tumour can be taken away by lateral thoracotomy and laminectomy may be necessary to deal with the remainder (Fig. 28). Which of these operations should be performed first depends upon

*Compression
of spinal cord*

whether or not signs of cord compression are present. If the tumour can be totally removed before paralysis has occurred the prognosis is good.

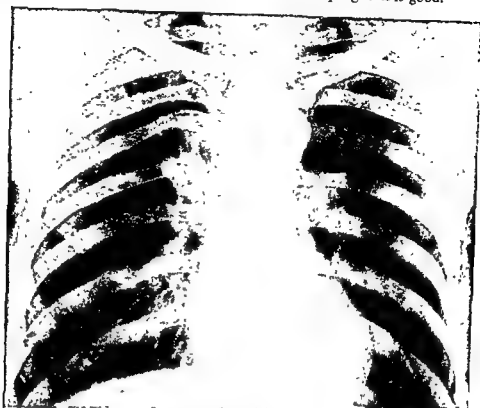


FIG. 28 —Dumb-bell neurofibroma. Skiagram of the chest of a patient showing a neurofibroma. The tumour was growing from an intercostal nerve and was partially disposed inside the spinal canal. The thoracic part was removed by lateral thoracotomy and the remainder by laminectomy. The patient returned to active duty in the Royal Navy.

(d) *Tumours of the sympathetic system*

These are rarer than the other types and are of four chief varieties. Embryonic tumours, called sympathogonia, sympathoblastoma, neuroblastoma and so on, are highly malignant; they usually occur in the upper part of the chest of children, and not only spread to the adjacent lymph glands but also invade ribs. Pain, debility and signs due to metastases are predominant; radiotherapy sometimes produces relief of symptoms but surgical treatment avails nothing.

Ganglioneuroma is the usual type; it is formed of adult nerve elements, occurs at any age and reaches any size. Large tumours are apt to degenerate and to liquefy centrally. The diagnosis of a mediastinal tumour is generally made radiologically, and the treatment is surgical removal by lateral thoracotomy. Neurofibromas occur in the supporting elements (that is, neuroglia) of the sympathetic system and have the same features as the solitary neurofibromas. Chromaffin tumours occur, but are very rare in the thorax.

All these tumours lie in the posterior mediastinum or in the paravertebral gutter, and are more often found in the upper than in the lower chest. Many are asymptomatic at first but if possible all should be removed surgically. The presence of pressure symptoms does not necessarily mean malignant change but invasion of ribs or of the brachial plexus is a sign of inoperability.

*Embryonic
tumours*

*Ganglio-
neuroma*

Neurofibroma

*Chromaffin
tumours*

*Compression
of spinal cord*

whether or not signs of cord compression are present. If the tumour can be totally removed before paralysis has occurred the prognosis is good.



FIG. 28.—Dumb-bell neurofibroma. Skigram of the chest of a patient showing a neurofibroma. The tumour was growing from an intercostal nerve and was partially disposed inside the spinal canal. The thoracic part was removed by lateral thoracotomy and the remainder by laminectomy. The patient returned to active duty in the Royal Navy.

(d) *Tumours of the sympathetic system*

*Embryonic
tumours*

These are rarer than the other types and are of four chief varieties. Embryonic tumours, called sympathogonia, sympathoblastoma, neuroblastoma and so on, are highly malignant; they usually occur in the upper part of the chest of children, and not only spread to the adjacent lymph glands but also invade ribs. Pain, debility and signs due to metastases are predominant; radiotherapy sometimes produces relief of symptoms but surgical treatment avails nothing.

*Ganglion-
neuroma*

Ganglioneuroma is the usual type; it is formed of adult nerve elements, occurs at any age and reaches any size. Large tumours are apt to degenerate and to liquefy centrally. The diagnosis of a mediastinal tumour is generally made radiologically, and the treatment is surgical removal by lateral thoracotomy. Neurofibromas occur in the supporting elements (that is, neuroglia) of the sympathetic system and have the same features as the solitary neurofibromas. Chromaffin tumours occur, but are very rare in the thorax.

Neurofibroma

*Chromaffin
tumours*

All these tumours lie in the posterior mediastinum or in the paravertebral gutter, and are more often found in the upper than in the lower chest. Many are asymptomatic at first but if possible all should be removed surgically. The presence of pressure symptoms does not necessarily mean malignant change but invasion of ribs or of the brachial plexus is a sign of inoperability.

8. TUMOURS ARISING NEAR THE NECK OF THE FIRST RIB

The name "superior pulmonary sulcus tumour" was applied by Pancoast (1932) to a tumour growing at the apex of the chest, which he believed to be a specific entity causing a special syndrome. The name means nothing and the tumour is not specific, but custom perpetuates both the name and the idea.

The cupola of the lung projects into the neck through the circle of the first rib and the mediastinum, and within this limited space important structures pass upwards and downwards. The first dorsal nerve and the sympathetic chain cross the neck of the rib; the first and second intercostal nerves together with the recurrent laryngeal and phrenic nerves are close by; any primary or secondary tumour growing in this space can involve these structures and so produce Pancoast's syndrome. Most of the tumours are pulmonary in origin and, being peripheral in the lung, grow outwards by direct extension into the brachial plexus, causing pain in the shoulder, pain down the arm and Horner's syndrome. Exactly similar effects can be produced by a primary or secondary tumour in the mediastinum if it lies near the neck of the first rib.

Many of the patients are treated at first as suffering from "neuralgia" and by the time the lesion is judged to be sufficiently serious to warrant radiography the ribs are already eroded. The chief complaint is of pain which steadily increases in intensity and for which neither radiotherapy nor surgery offers any hope of alleviation or cure. The tumours cannot be removed, and operations performed to relieve the pain, such as division of the relevant intercostal nerves or even of the brachial plexus itself, are not successful. It is debatable whether cordotomy is justifiable in such cases.

REFERENCES

- Allison, P. R. (1947). *Thorax*, 2, 176.
 Barnard, W. G. (1926). *J. Path. Bact.*, 29, 241.
 Barrett, N. R. (1939) *Tubercle, Lond.*, 20, 445.
 — (1946). *Thorax*, 1, 48.
 — and Thomas, D. (1944) *Brit. med J*, 2, 692
 Blades, B. (1946) *Ann. Surg.*, 123, 749.
 Brown, R. K., and Robbins, L. L. (1944) *J thorac Surg*, 13, 84
 Handley, R. (1947) *Lancet*, 2, 874.
 Harrington, S. W. (1929). *Arch. Surg, Chicago*, 19, 1679
 Mounsey, J. P. D. (1947). *Thorax*, 2, 203.
 Neuhoff, H., and Jeremin, E. E. (1943). *Acute Infections of the Mediastinum*. Baltimore; Williams & Wilkins
 Olsen, A. M., and Wilson, G. T. (1944). *J thorac. Surg*, 13, 53
 Pancoast, H. K. (1932). *J. Amer. med Ass.*, 99, 1391.
 Phillips, C. E. (1938) *J. Amer. med Ass.*, 111, 998.
 Rusby, N. L. (1944). *J. thorac. Surg.*, 13, 169.
 Sanes, M. D., MacManus, J. E., and Scratchard, G. N. (1945) *J. thorac Surg*, 14, 253
 Smart, J., and Thompson, V. C. (1947). *Thorax*, 2, 163.
 Tubbs, O. S. (1946). *Thorax*, 1, 247.

[References to other titles are given under Heart, Mediastinum, Myasthenia Gravis, and Thymus, in the Index Volume. The subject is also dealt with under the heading of Mediastinum Diseases in the *British Encyclopaedia of Medical Practice* (1938), Vol. 8, p. 438.]

MELAENA AND BLOOD IN THE STOOLS

BY EDWARD R. CULLINAN, M.D., F.R.C.P.
PHYSICIAN, ST. BARTHOLOMEW'S HOSPITAL; PHYSICIAN, WESTMINSTER
(GORDON) HOSPITAL, LONDON

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1. INTRODUCTION

231.] *The discovery of blood in the stools, whether visible or occult, should always be considered a serious finding. With the exception of fissures and piles, there are few, if any, trivial or innocuous causes.*

2. AETIOLOGY

Blood in the stools may result from extrinsic disorders outside the gut, such as circulatory failure and the blood dyscrasias. Until proved otherwise, however, it should be regarded as evidence of an intrinsic lesion of the gut itself.

3. DIAGNOSIS

The following paragraphs refer to blood in the stools resulting from the more important of these intrinsic lesions. It is not proposed to discuss the detailed differential diagnosis or to enumerate all the possible causes. In this respect it should be remembered, when searching for a diagnosis, that the commonest cause is the most likely one. The possible causes of blood in the stools are many; the probable causes in a particular case are few. In many instances special pathological, sigmoidoscopic and radiological investigations will be required to establish a diagnosis. In no instance will these special investigations make up for the lack of an accurate history, and a detailed clinical examination of the patient, his rectum and his stools.

The simplest and surest way of knowing that there is visible blood in the stools is to look at them. (The white surface of the bed-pan makes an excellent background.) Too often this simple observation is neglected, and the patient's own story is relied upon. The story is often quite valueless. A patient may not notice that he has passed a large melaena stool, and is even less likely to notice one which is just streaked with blood. By looking at the stool one can see more than the presence of blood. One can notice its colour, whether or not it is intimately mixed with the faeces, the consistency of the faeces and the added presence of pus and mucus. This personal inspection of the stools should never be omitted. *Inspection of stools*

Then again, knowledge that occult blood is present in the stools is of great importance. The test for occult blood, however, is often neglected. For general use the test with benzidine is too sensitive, and the pyramidon test is the best for ordinary purposes.

Pyramidon test

The patient is kept on a meat-free diet for 3 days, during which time it is best for him not to use a tooth-brush. He should be examined for the possibility of bleeding piles.

The stools are collected in any clean container which is available, and a portion the size of a plum stone is emulsified in 3 or 4 millilitres of water. A few drops of 33 per cent acetic acid are added to the specimen which is then boiled, preferably in a water-bath, for 5 minutes, and then allowed to cool. To approximately 2 millilitres add 1 millilitre of 5 per cent alcoholic solution of pyramidon, superimposed as a layer. Drop in 6 drops of hydrogen peroxide with a splash. If occult blood is present, a mauve ring will appear spreading up into the pyramidon. The amount of blood which is required to give a positive reaction is uncertain: probably one drop is sufficient. It is customary to test 3 successive stools on 3 successive days.

4. MELAENA STOOLS

(μ$\epsilon\lambda\alpha\iota\alpha\ =\ \text{black-bile}$)

A melaena stool is very dark in colour and tarry in consistency, usually unformed and semi-solid, and frequently offensive. It is more sticky than a stool which has been blackened by iron, bismuth or charcoal and it tends to adhere to the side of the pan. Only when the stool is semi-solid is it black and tarry. When it is loose, the colour tends to be cherry-red. If part of a melaena stool is mixed with twice its volume of water and allowed to stand, the water becomes reddish in colour.

A melaena stool is caused by blood coming from high up in the alimentary tract. The site of bleeding is almost always higher than the jejunum, but very occasionally may be lower in the small intestine. Blood from a gastro-jejunal ulcer may give rise to a melaena stool, or it may be recognized from its colour as frank blood. It is said that a melaena stool can result from the presence of as little as 2 ounces of blood. It usually results from a massive haemorrhage, by far the commonest cause of which is peptic ulceration of the stomach or duodenum. A single large haemorrhage may give rise to one or more melaena stools which may not appear until several days later. Whether or not the haemorrhage is followed also by haematemesis does not necessarily alter the significance of melaena stools. Haematemesis depends upon several factors, notably on the speed with which the stomach fills with *Site of bleeding*

blood. In most instances it is fairly easy to decide when a patient has had a gross internal haemorrhage, but proof that it has been caused by bleeding from a peptic ulcer is sometimes impossible to obtain and the diagnosis may have to be inferred.

(1) Peptic ulcer

Persistence of pain

About 25 per cent of patients suffering from peptic ulcer have haemorrhage as the first symptom. Moreover, when typical ulcer pain has been present before a haemorrhage, the pain usually disappears immediately after the haemorrhage. Indeed, the persistence of pain usually portends a grave outcome. Nevertheless, the diagnosis will depend largely upon the history. Examination of the patient should be sufficiently thorough to exclude other general causes of haemorrhage, but palpation of the abdomen must be very gentle.

An estimation of the haemoglobin concentration immediately after a haemorrhage may conceal the gravity of the bleeding. Only after the blood has been diluted—and this may take a day or so—will the reading give a true indication of the degree of blood loss.

Periodic massive haemorrhages

An x-ray examination of the stomach and duodenum should not be undertaken until 3 weeks after bleeding has stopped and, even then, the radiologist should be warned of what has happened. Sometimes the skiagram does not reveal the source of bleeding and, in some patients who die as a result of such a haemorrhage, no lesion can be found in the stomach or duodenum at post-mortem examination. In such patients it is generally assumed that the bleeding has originated from an acute ulcer or an erosion which has healed. One seldom sees nowadays brisk haemorrhages from acute peptic ulcers in young women, as were described in the early part of the century, but all are familiar with the adult male who has periodic massive haemorrhages, presumably from the duodenum, with few intervening symptoms and no radiological evidence of ulceration.

Early gastroscopy after massive haemorrhage is too hazardous an undertaking for any but the most expert, working in ideal hospital conditions.

(2) Portal hypertension

In approximately 4 per cent of patients who have melaena stools, the cause is portal hypertension resulting usually from cirrhosis of the liver, less often from Banti's syndrome, and very rarely from portal thrombosis. In the great majority of these the bleeding occurs from varices in the lower part of the oesophagus. Only very occasionally can these varices be demonstrated radiologically.

(3) Gastric carcinoma

Melaena stools are uncommon in gastric carcinoma. When they do appear, the disease is usually well advanced and there is a history of previous gastric symptoms and of progressive loss of weight.

(4) Other causes

Swallowed blood

Swallowed blood may have to be considered, arising either from the lungs, the pharynx or the nose. Epistaxis may not always be obvious when it happens during sleep and the blood is swallowed.

Other causes of melaena stools are rare. They include inflammation, ulceration, and malignant or benign tumours of the oesophagus; ulceration in a hiatus hernia of the stomach; rupture of an arteriosclerotic vessel; trauma; benign tumours of the stomach (leiomyomas) or of the duodenum, in which haemorrhage may be the initial symptom; hereditary haemorrhagic angiomatosis; local malignant tumours eroding the stomach or duodenum, or even the rupture of an aneurysm.

5. FRANK BLOOD VISIBLE IN THE STOOLS

(1) From the small intestine

Severe bleeding may take place in the small intestine during the later stages of typhoid or paratyphoid fever. Mesenteric thrombosis or embolism is followed by the passage of blood in the stools, the colour depending upon the site of the arterial occlusion. Frank blood visible in the stools resulting from a lesion of the small intestine, especially in the absence of other florid symptoms or signs, is unusual. It is rare in tuberculous enteritis, although occult blood is often present. In regional ileitis it is seldom seen unless the colon is also involved. Although tumours may cause gross haemorrhage, they are uncommon in the small intestine. Only 3 per cent of malignant growths of the gastro-intestinal tract occur in this part of the gut. Benign tumours such as myomas and adenomas are rare and fibromas, lipomas and angiomas are very rare.

In children, particularly in boys, the sudden passage of blood, bright or dark, in the stools, is suspicious of peptic ulceration in a Meckel's diverticulum. Other physical signs may be absent, and only sometimes has there been a preceding history of abdominal pain 1-2 hours after food. Adenomatous polyps in the lower gut are an occasional source of bleeding in children, although a blood-stained discharge from the rectum is more usual than massive haemorrhage. The polyps can be recognized by means of rectal examination and sigmoidoscopy.

Intussusception in infants may produce blood-stained mucus, looking like red-currant jelly, which appears on the napkin within a few hours of the onset of pain. It may, at first, be mixed with faeces. In the rare form of intussusception involving the jejunum, the stool is said to look like melted strawberry ice. Blood and mucus may be present in the stools of infants suffering from acute gastro-enteritis, but usually follows a simple diarrhoea.

(2) From the large intestine and the rectum

In general, blood in the stools from the proximal part of the large bowel is darker and more intimately mixed with faeces than is that from the distal part. For example, blood from the pelvic colon or rectum may be quite bright, unclotted and not mixed with the faecal masses. It should be remembered, however, that if the stool is unformed, blood mixed with faeces may originate from the rectum as well as from higher up. Haemorrhages from the rectum or from the anal canal may streak the outer surface of the stool, or blood may follow the stool, spattering the pan. In such cases, however, a lesion in the sigmoid colon should always be excluded.

A soft or fluid stool containing mucus, blood and pus is evidence of ulceration of the large intestine.

(3) Causes of blood-stained stools**(a) The dysenteries***Acute
bacillary
dysentery*

In warm countries the diagnosis of acute bacillary dysentery caused by *Bacillus dysenteriae* Flexner and *B. dysenteriae* Shiga will seldom present much difficulty. At first stools consist in glairy blood-stained mucus with little or no faecal material, and there may be as many as 40 stools a day. Later, there is more faecal material and the stools become offensive.

B. dysenteriae Sonne is the common pathogen of bacillary dysentery in the United Kingdom. Usually the stools are soft or fluid and contain varying quantities of visible blood, but the appearances are often inconstant and misleading. The diagnosis may easily be missed unless this disease is thought of, and will always remain in doubt until bacteriological examination confirms it.

*Amoebic
dysentery*

Amoebic dysentery is another disease which may be difficult to recognize, since attacks are often so insidious and mild. Characteristically, the stools are soft or fluid with much or little blood and mucus and a little pus, and have an offensive smell. Stools should be examined for *Entamoeba histolytica* within 10 minutes of being passed into a warm bed-pan, or, best of all, by immediate microscopy of material obtained by swabbing the ulcers through the sigmoidoscope.

(b) Idiopathic ulcerative colitis

When idiopathic ulcerative colitis involves the whole colon the stools are fluid and foul. They contain blood, pus and mucus intimately mixed with the faeces. When the disease process is confined to the distal colon, the blood is less well intermingled with the faeces but may be profuse. Sometimes, in this distal type, there is no true diarrhoea. Many motions will still be passed each day but most of them will consist only of blood and mucus. There may even be constipation, with blood and mucus following the constipated stool or covering its surface. Even then, there are usually, in addition, frequent intermediate motions of blood and mucus.

*Sigmoidoscopic
appearances*

The sigmoidoscopic appearances may be similar whether the disease affects the whole colon or only the distal part, because with rare exceptions the brunt of the disease in either type falls on the upper rectum and lower sigmoid colon.

(c) Other infections and infestations*Rectal
bilharziasis*

In certain countries rectal bilharziasis, caused by infestation with *Schistosoma mansoni*, may have to be considered. The site of the lesion is in the rectum and bleeding is a common symptom. Diagnosis depends upon the discovery of the eggs in the stools; a specimen is best obtained by means of a rectal swab. Eggs are seldom found less than 3 months after infestation.

Rectal bleeding in small amounts, recurring almost daily for months, may be caused by lymphopathia venereum, long before a stricture develops. The proctoscopic appearances are not characteristic and diagnosis will usually depend upon pathological findings, particularly the result of the Frei test.

(d) Cancer

In the absence of a proven colitis or proctitis, either infective or idiopathic, the presence of frank blood in the stools of an adult should always mean carcinoma until proved otherwise. It may be the earliest and only sign.

A change of bowel habit, alternating diarrhoea and constipation, or a few loose stools in the morning are all suspicious symptoms. Depending upon the site and state of the lesion, the appearances of the stools vary. In some instances there may be only a little blood and mucus on the faecal surface; in others there may be blood, pus and mucus in a solid or liquid stool, highly suggestive of an ulcerating growth. Immediate and thorough investigation is required. *Varied types of stools*

It is not proposed here to discuss haemorrhoids and fissures which are such a frequent cause of bleeding, but it must be repeated that they should not be accepted as the origin of blood in the stools until a growth has been excluded. Every patient with piles must be examined to see whether he has cancer of the rectum. A large number of cancers of the colon and rectum are within reach of the examining fingers, and more within reach of the sigmoidoscope. The remainder may be confirmed by x-ray examination.

(e) Other lesions

Polypi, polyposis and chronic diverticulitis may all produce considerable quantities of blood of varying shades of red. The finding of polypi should raise the suspicion that cancer is present. The radiological appearances of diverticulitis in the distal colon should be accepted with reserve and, even if it is decided that the lesion is not malignant, the patient should be kept under observation, x-ray examinations being repeated from time to time. *Necessity for x-ray examination*

Hard constipated stools may irritate the mucosa and cause brief attacks of diarrhoea. These may be accompanied by a little blood, especially if the anus is tight. The diagnosis should never be made until the more serious possibilities have been dismissed.

6. OCCULT BLOOD IN THE STOOLS

Any of the causes already mentioned may produce occult blood in the stools. The majority of cancers in the digestive tract bleed, and the test for occult blood is often of great diagnostic importance. In an adult, if there are gastric symptoms and occult blood is constantly present, carcinoma of the stomach is probable. The absence of occult blood does not exclude the diagnosis. Again, the finding of occult blood may lead to the diagnosis of a cancer of the colon but its absence does not exclude such a diagnosis. *Test for occult blood important*

7. CONCLUSION

Whatever other investigations are made for the diagnosis of gastro-intestinal disease, the stools must always be examined.

[References to other titles are given under Melaena and Blood in the Stools in the Index Volume.]

MELANOMA

BY GEORGIANA M. BONSER, M.D., M.R.C.P.
BROTHERTON FELLOW IN CANCER RESEARCH, UNIVERSITY OF LEEDS

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1. DEFINITION AND CLASSIFICATION

232.] The tumours of this group are characterized by the formation of an iron-free brown pigment, melanin, which is derived by oxidation from a colourless precursor. The following classification is based on morbid histology and ontogeny.

Neurogenic	{ benign melanoma malignant melanoma }	{ cutaneous ocular }
Epidermic	{ melanin-forming squamous papilloma melanin-forming basal-cell cancer melanin-forming squamous cancer melanin-forming benign calcified epithelioma }	

It is essential to separate the neurogenic and epidermic groups because of differences in prognosis and treatment.

2. NOMENCLATURE

A precise terminology is desirable. Terms such as "naevus" (which is also applied to vascular and lymphangiomatous tumours of the skin) and "mole" are lacking in precision. The latter term is usually applied only to neurogenic melanomas, and may be adjectivally qualified as simple or malignant and pigmented or non-pigmented. "Melanoma" should be reserved for the neurogenic group, always bearing in mind that "non-pigmented melanomas" occur. The epidermic varieties are best designated simply as melanin-forming papillomas or basal-cell or squamous-cell carcinomas, as the case may be.

3. MORBID ANATOMY AND HISTOLOGY

(I) Cutaneous benign melanoma

Many of these tumours are congenital but they may appear at any age. The colour range is from pale buff to dark brown, but occasionally melanin is

mpletely lacking (so-called non-pigmented mole). If the pigment is situated deep in the corium or beneath it, a blue colour is apparent (so-called blue naevus). The surface may be smooth or rough, hairless or hairy, and the tumour is more likely than not to project slightly from the surface. It has a complete covering of epidermis; ulceration, unless due to some known cause, usually indicates malignancy or a move in that direction.

The characteristic cells are the "naevus" cells or melanoblasts, which are polygonal cells lying in groups either in the dermis or immediately subjacent to the epidermis. These pigment-forming cells, as shown by Masson (1926), originate in association with a complicated nerve plexus in the dermis, derived from the sheath of the

tactile end-organ; indeed it is not uncommon to find frank neurofibromatous elements in the deeper part of the tumour. Other authors, notably Dawson (1925),

have regarded the naevus cell as being of epidermic origin, but it is to be noted that the tumours show no tendency towards epidermal evolution.

(2) Cutaneous malignant melanoma

This tumour may arise in a pre-existing benign melanoma (Fig. 29) or *de novo* in previously healthy skin. Malignancy is indicated by rapid growth, spread, rapidly increasing depth of pigmentation, and ulceration. The tumour spreads both marginally and deeply, besides projecting from the surface (Fig. 30). Dissemination usually takes place first into the regional lymph glands; later there is blood-borne metastasis to the liver and other organs.

Microscopically, the "naevus" cells show evidence of great proliferative activity. They may retain their polygonal form, but they tend to be larger and less regular, with large, irregular and hyperchromatic nuclei, and melanin is usually present in abundance. In general they still retain their lepidic (Adam, 1910)



Appearance

Histology

FIG. 29.—Malignant melanoma arising in a soft brown benign melanoma of skin of breast in a woman aged 41 years. The benign tumour had been present and growing for 3 years when the malignant nodule appeared. Excision was performed. The patient died 4 years later of general dissemination of malignant melanoma (Natural size)



FIG. 30.—Projecting malignant melanoma of forearm in a woman aged 72 years. This tumour had first appeared 18 months previously and had grown rapidly in recent months, causing ulceration of the surface. Note the sharply demarcated black and white areas (Natural size.)

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This tumour may arise in a pre-existing benign melanoma (Fig. 29) or *de novo* in previously healthy skin. Malignancy is indicated by rapid growth, spread rapidly increasing depth of pigmentation, and ulceration. The tumour spreads both marginally and deeply, besides projecting from the surface (Fig. 30). Dissemination usually takes place first into the regional lymph glands; later there is blood-borne metastasis to the liver and other organs.

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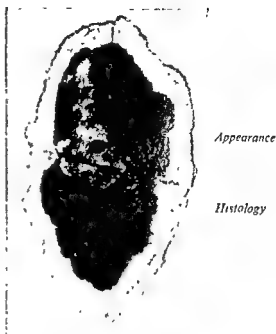


FIG. 29 — Malignant melanoma arising in a soft brown benign melanoma of skin of breast in a woman aged 41 years. The benign tumour had been present and growing for 3 years when the malignant nodule appeared. Excision was performed. The patient died 4 years later of general dissemination of malignant melanoma. (Natural size.)

MELANOMA

BY GEORGIANA M. BONSER, M.D., M.R.C.P.
BROTHERTON FELLOW IN CANCER RESEARCH, UNIVERSITY OF LEEDS

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1. DEFINITION AND CLASSIFICATION

232.] The tumours of this group are characterized by the formation of an iron-free brown pigment, melanin, which is derived by oxidation from a colourless precursor. The following classification is based on morbid histology and ontogeny.

Neurogenic	{ benign melanoma malignant melanoma }	{ cutaneous ocular }
Epidermic	{ melanin-forming squamous papilloma melanin-forming basal-cell cancer melanin-forming squamous cancer melanin-forming benign calcified epithelioma }	

It is essential to separate the neurogenic and epidermic groups because of differences in prognosis and treatment.

2. NOMENCLATURE

A precise terminology is desirable. Terms such as "naevus" (which is also applied to vascular and lymphangiomatous tumours of the skin) and "mole" are lacking in precision. The latter term is usually applied only to neurogenic melanomas, and may be adjectivally qualified as simple or malignant and pigmented or non-pigmented. "Melanoma" should be reserved for the neurogenic group, always bearing in mind that "non-pigmented melanomas" occur. The epidermic varieties are best designated simply as melanin-forming papillomas or basal-cell or squamous-cell carcinomas, as the case may be.

3. MORBID ANATOMY AND HISTOLOGY

(1) Cutaneous benign melanoma

Many of these tumours are congenital but they may appear at any age. The colour range is from pale buff to dark brown, but occasionally melanin is

completely lacking (so-called non-pigmented mole). If the pigment is situated deep in the corium or beneath it, a blue colour is apparent (so-called blue naevus).

The surface may be smooth or rough, hairless or hairy, and the tumour is more likely than not to project slightly from the surface. It has a complete covering of epidermis; ulceration, unless due to some known cause, usually indicates malignancy or a move in that direction.

The characteristic cells are the "naevus" cells or melanoblasts, which are polygonal cells lying in groups either in the dermis or immediately subjacent to the epidermis. These pigment-forming cells, as shown by Masson (1926), originate in association with a complicated nerve plexus in the dermis, derived from the sheath of the

facile end-organ; indeed it is not uncommon to find frank neuro-fibromatous elements in the deeper part of the tumour. Other authors, notably Dawson (1925),

have regarded the naevus cell as being of epidermic origin, but it is to be noted that the tumours show no tendency towards epidermal evolution.

(2) Cutaneous malignant melanoma

This tumour may arise in a pre-existing benign melanoma (Fig. 29) or *de novo* in previously healthy skin. Malignancy is indicated by rapid growth, rapidly increasing depth of pigmentation, and ulceration. The tumour spreads both marginally and deeply, besides projecting from the surface (Fig. 30). Dissemination usually takes place first into the regional lymph glands; later there is blood-borne metastasis to the liver and other organs.

Microscopically, the "naevus" cells show evidence of great proliferative activity. They may retain their polygonal form, but they tend to be larger and less regular, with large, irregular and hyperchromatic nuclei, and melanin is usually present in abundance. In general they still retain their lepidic (Adami 1910)



Appearance

Histology

FIG 29.—Malignant melanoma arising in a soft brown benign melanoma of skin of breast in a woman aged 41 years. The benign tumour had been present and growing for 3 years when the malignant nodule appeared. Excision was performed. The patient died 4 years later of general dissemination of malignant melanoma (Natural size)



FIG. 30.—Projecting malignant melanoma of forearm in a woman aged 72 years. This tumour had first appeared 18 months previously and had grown rapidly in recent months, causing ulceration of the surface. Note the sharply demarcated black and white areas (Natural size.)

Spread

Histology

or rind character—hence the term, melanotic carcinoma—or to assume the structure of a spindle-cell sarcoma.

As the tumour enlarges, necrobiotic changes become prominent. pigment liberated from the disintegrating tumour cells is taken up by phages, which may become very numerous in and around the areas of

(3) Ocular melanomas

These tumours originate most frequently in the choroid coat but may also in the conjunctiva, iris or ciliary body. A benign type is described (Elder, 1940) but the malignant type is more commonly seen. In distinction to their cutaneous counterpart, malignant melanoma of the uveal tract tend to be of sarcomatous or hyaline type. They are more likely to disseminate by the blood stream, but they are also capable of great destructive effect on the eyeball and they invade the orbit by direct extension. In some instances they spread along the sheath of the optic nerve and so extend into the pia-arachnoid of the brain and cord.

(4) Melanin-forming epidermal tumours

This group is well recognized in some European countries, notably in the United States of America, but it has received scant attention in Britain. In clinical behaviour and histological features (except for the content) these tumours are similar to the corresponding non-pigment-producing skin tumours; that is to say they are either simple squamous papillomas or basal-cell, intra-epidermic or frankly malignant squamous carcinomas (Stewart and Bonser, 1948). A good many occupy a position intermediate between the basal and squamous-cell types (transition forms). It cannot be any question that in these tumours the melanin is formed in

the neoplastic cells. Its content varies greatly, and in the most heavily pigmented tumours much of it is contained within macrophages in the intercellular spaces, especially in the papillae of the true skin.



FIG. 31.—Pedunculated malignant melanoma arising in a soft brown benign melanoma of the back in a man aged 73 years. Note the uneven depth of pigmentation and compare with Fig. 36. (Natural size)

4. CLINICAL PICTURE

(1) Incidence of melanoma

Most people are able to develop one or more benign cutaneous melanomas, which remain benign throughout life. As melanoma is a rare tumour, not all these tumours arise. Some pre-existent benign growths may change from the benign to the malignant. A corresponding rare one. When it does occur, it is thought to be more common in the soft brown melanoma

in the hairy mole (Figs. 29 and 31). Benign melanomas occur in any site, the *Benign* order of frequency being the face and neck, the trunk and then the extremities (excluding the palms of the hands and the soles of the feet). An extensive,



FIG. 32.—Ulcerating malignant melanoma of the heel. This was an extensive, deeply pigmented tumour encircling the heel in a man aged 47 years. It had been present and growing for 4 years. There were already widespread metastases in regional lymph glands, brain, liver and other organs. (Half natural size.)



FIG. 33.—Subungual malignant melanoma of the thumb in a man aged 74 years. Ten years previously he had been wounded at this site by a spark from an anvil. The wound was excised a year later and it healed. Seven and a half years later, there was a recurrence of the sore due to the pulling down of a piece of rough skin at the nail-bed. The melanoma developed at this site, and when the patient was examined there were already meta-
Malignant stases in the axillary glands. The right figure shows the lateral aspect of the thumb with the tumour at the top. The left figure shows a longitudinal section, demonstrating the encircling of the nail-bed by the tumour. (Half natural size.)

hairy, "bathing-drawers" type is described.

Malignant melanomas occur more frequently on the extremities (Fig. 32) than on the face and neck (Affleck, 1936), the nail-bed (subungual) being a well-recognized site (Fig. 33). They also occur in the vulvar region. It is suggested by many authors that trauma accounts for this distribution.

Evidence of malignant change in a pre-existing benign tumour consists in deepening of the pigmentation, enlargement, ulceration, the appearance of satellite tumours around the primary one, or absence of change in the primary tumour but the presence of enlarged regional lymph glands.

(2) Dissemination

Dissemination of malignant melanoma, whether cutaneous or ocular, is a curiously variable, erratic process. In a few cases dissemination by the blood

Blood stream

Lymphatic spread

General metastasis



FIG 34 — Metastases of malignant melanoma in the kidney (Half natural size)

retrogression of metastases is described in rare cases. (Willis, 1934.)

(3) Ocular melanomas

The ocular melanomas may be seen in their various sites upon examination of the eye (Fig. 35). Benign tumours are usually small and occur most commonly in the choroid. Whenever glaucoma supervenes in an eye which is the seat of a benign melanoma, malignant change should be

Glaucoma and malignancy



FIG 35.—Malignant ocular melanoma arising in the iris and causing detachment of the retina. (Natural size.)

suspected (Duke-Elder, 1940)

Malignant ocular melanomas may be symptomless at first, but glaucoma, extra-ocular extension and metastases supervene after varying intervals of time.

(4) Epidermic tumours

Pigment-forming squamous papillomas or hard moles (also called pigmented acanthomas) are usually irregular, finely nodular, warty masses, which may be sessile or pedunculated. They vary in colour, but the surface keratin usually masks the underlying pigment and gives the tumours a dirty pig or slaty colour.



FIG 36 — Melanin-forming basal-cell cancer of the temple in a man aged 68 years. Note the sessile tumour, half above and half below the epidermic level with areas of brown pigmentation on the cut surface. Compare with Fig 31. (Natural size)

Pigmented basal-cell cancers present all the characteristics of their non-pigmented counterparts except for their colour (Fig. 36). Thus a cicatrized floor surrounded by a beaded margin is often described (Touraine, 1935). The depth of colour is usually irregular—black, brown and grey areas being present in the same tumour. Many, however, are more uniformly slaty. The history of this group of tumours is usually very long. Not infrequently they have been present for 10 years or more and have grown gradually and progressively. They are more common on the face and neck than in other sites. *Site* *Basal-cell cancers*

5. DIAGNOSIS

The only certain method of diagnosis is by histological examination, which is necessary to differentiate both the neurogenic from the epidermic type and the benign from the malignant. It is often possible to make a tentative diagnosis of the epidermic pigmented tumours from the clinical appearance, history, rate of growth and so forth.

6. PROGNOSIS

This is entirely dependent upon the histological diagnosis. A benign cutaneous melanoma, completely excised, does not recur; a malignant melanoma, however radically excised, is highly likely to recur or to metastasize with or without local recurrence, although this process may be delayed for periods up to many years. The prognosis of the malignant melanoma, whether ocular or cutaneous, is usually regarded as very bad, although radical excision with dissection of regional glands yields fairly satisfactory results in some hands (Bickel, Meyerding and Broders, 1943). A pigmented squamous papilloma is cured by radical excision; pigmented basal-cell cancers do not metastasize and they respond well to excision or to irradiation therapy.

7. INDICATIONS FOR SURGICAL INTERVENTION

There is much difference of opinion on this point, which is discussed by Tod (1944). For example, the patient may ask for the cosmetic removal of a pigmented mole (benign melanoma), which has been present for a long time and shows no sign of growth. Some surgeons would advise strongly against this, but all would agree that if the patient insists the tumour should be removed radically. Other authors advocate the prophylactic removal of pigmented cutaneous tumours, especially in sites subject to trauma or irritation (Affleck, 1936). *Benign melanomas*

By contrast, the patient may seek advice because a pigmented lesion has started to grow spontaneously, after injury or after injudicious treatment. In these cases, surgical treatment must be radical, "the fear that repair may present difficulties must not influence the surgeon in planning his excision" (Tod, 1944). Whenever the regional lymph glands are enlarged, the primary tumour and the glands must be widely excised, preferably with block dissection of the intervening lymphatics. When multiple metastases are present, palliative radiotherapy is usually advised, although these tumours are known to be radio-resistant. The successful treatment of pigmented basal-cell cancers by radiotherapy accounts for some of the cures claimed. *Malignant melanomas*

Benign ocular melanomas should be left alone. Once a malignant tumour is suspected, enucleation should be performed with removal of as much posterior orbital tissue as possible including a maximal length of optic nerve.

REFERENCES

- Adami, J. G. (1910). *Principles of Pathology*, 2nd ed. Philadelphia and New York; Lea & Febiger. London; Oxford University Press.
- Affleck, D. H. (1936). *Amer. J. Cancer*, 27, 120.
- Bickel, W. H., Meyerding, H. W., and Broders, A. C. (1943). *Surg. Gynec. Obstet.*, 76, 570.
- Dawson, J. W. (1925). *Edinb. med. J.*, 32, 509.
- Duke-Elder, W. S. (1940). *Text-book of Ophthalmology*, Vol. 3, p. 2477. London; Kimpton.
- Gleave, H. H. (1929). *Lancet*, 2, 658.
- Masson, P. (1926). *Ann. Anat. path. méd-chir.*, 3, 417 and 657.
- Stewart, M. J., and Bonser, Georgiana M. (1948). *J. Path. Bact.*, 60, 21.
- Tod, Margaret C. (1944). *Lancet*, 2, 532.
- Touraine, A. (1935). *Ann. Derm. Syph., Paris*, (7th series), 6, 785.
- Willis, R. A. (1934). *The Spread of Tumours in the Human Body*. London; Churchill.
- [References to other titles are given under Melanoma in the Index Volume. The subject is also dealt with under the heading of Skin Diseases: Tumours in the *British Encyclopaedia of Medical Practice* (1939), Vol. 11, p. 200.]

MENINGES—MENINGITIS, ACUTE AND CHRONIC

By G. L. ALEXANDER, B.Sc., F.R.C.S.

DIRECTOR, NEUROSURGICAL UNIT, UNIVERSITY OF BRISTOL; FORMERLY
ASSISTANT NEUROLOGICAL SURGEON, ROYAL INFIRMARY, EDINBURGH

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1. INTRODUCTION

233.] The subject of meningitis cannot logically be discussed without consideration of infective thrombophlebitis of the brain and of subdural abscess, and therefore those conditions receive attention in this chapter.

2. SURGICAL ANATOMY AND PHYSIOLOGY

The dura mater is the most external of the three layers of meninges, and its deep aspect is everywhere in contact with the arachnoid mater, separated from it only by a potential space wherein subdural pus may accumulate.

The subarachnoid space lies between the arachnoid and the pia mater, *The meninges* invests the central nervous system and contains cerebrospinal fluid. The space is traversed by innumerable arachnoid trabeculae and is lined throughout by cells of "endothelial" type. The larger arteries and veins subserving the neuraxis also traverse it. The pia mater is virtually integral with the brain, spinal cord and nerve roots.

The subarachnoid space is fed with fluid which is formed within all four ventricles by selective transudation from the choroid plexuses. The fluid

circulates within the subarachnoid space, ascends around the brain-stem, into the fissures and sulci and is transmitted into the venous blood stream at the Pacchionian villi in relation to the superior longitudinal and lateral sinuses. The main fluid channels on the surface of the brain follow the course of the main arteries and the circulation of cerebrospinal fluid is in part promoted by their pulsation. Tapering sleeve-like prolongations of the subarachnoid space surround the smaller arteries as they penetrate the brain. These perivascular spaces (Virchow-Robin) are important because they facilitate interchange of diffusible substances and protein between the brain and the cerebrospinal fluid, and because the infection of meningitis is not, in fact, confined to the surface of the brain but, from the outset, extends into its substance—a superficial encephalitis. The direction of the spread of meningitis is influenced by the slow stream of fluid on the surface of the brain.

The normal cerebrospinal fluid contains one or two lymphocytes per cubic millimetre. The protein content is about 30 milligrams per 100 cubic centimetres (ventricular fluid, about 15 milligrams in 100 cubic centimetres); of chlorides there are about 720 milligrams, and of sugar, 50–60 milligrams in 100 cubic centimetres.

The normal lumbar fluid pressure in the horizontal position is about 80–180 millimetres of water. This pressure is not constant but varies considerably at rest in the normal subject. Coughing, holding the breath and abdominal compression raise the pressure temporarily to 250 millimetres or more.

3. PACHYMENINGITIS

This cannot be regarded clinically as a disease *per se*. It is true that the dura mater shares in the inflammatory changes accompanying infection in the tissues adjacent to it, but this tough, rather avascular membrane constitutes a valuable natural barrier which delays the spread of infection. When infection spreads from the exterior and through the dura it does so by way of small veins and thus, by continuity, thrombophlebitis of dural blood sinuses and of cerebral or cerebellar veins can be established; conversely, transdural spread from within outwards can sometimes cause extradural suppuration.

The spinal dura mater is occasionally thickened as a result of attenuated coccal infection in the epidural space. Spinal pachymeningitis is an occasional late sequel of typhoid fever. Tuberculous or syphilitic pachymeningitis is not rare. Clinically, spinal pachymeningitis is brought to notice when compression of the spinal cord eventuates, the mechanism being due either to the tumefaction of indolent granulations in the epidural space or to the stricturing effect of fibrosis of the theca.

4. ACUTE PYOGENIC LEPTOMENINGITIS

(1) Aetiology

Considered from the surgical aspect, acute leptomeningitis is usually a complication arising from infection in tissues adjacent to the meninges, as, for example, from the exterior in open fractures of the skull and from the interior by spread from an embolic brain abscess. Dissemination by the blood stream also occurs, and it is possible for infective foci to be established extracranially

Circulation
of cerebro-
spinal fluid

Normal
cerebrospinal
fluid

and intracranially at the same time, via the external and internal carotid arteries.

The various pyogenic cocci are encountered, the commonest being streptococci, pneumococci and staphylococci. Infections due to *Bacillus coli*, *B. influenzae* or *B. proteus* are rather less common. Recently, with the advent of penicillin, *B. pyocyaneus* has acquired importance; moreover, the danger of misplaced trust in "sterile" water for rinsing lumbar-puncture instruments is being realized. Rarer and occasional cases of infection due to *B. typhosus*, the actinomyces, gonococci or fungi may be encountered.

(2) Pathology

Routes of infection

Infection may be introduced into the subarachnoid space directly from the exterior by penetrating wounds of the head and spine, or by comminuted or fissure fractures of the skull implicating the nasal passages, paranasal air sinuses or middle ear, especially if operative treatment is inadequate or delayed. In health the air sinuses are sterile, but if blood clot therein becomes infected or if the sinuses are already diseased, the implications are obvious.

Wounds of head and spine

Even the simple scalp wound, if allowed to become septic, carries the danger of intracranial extension of infection by trans-osseous venous pathways.

Acute or chronic infection of the middle ear or paranasal sinuses is at any time apt to extend through the dura mater, usually by a spreading infective thrombophlebitis or, much less often, by direct irruption into the intracranial cavity. Thus brain abscess becomes a likely sequel.

Ear and nasal sinus infections

Infective thrombophlebitis of the brain is accompanied by an inflammatory reaction in the leptomeninges; conversely, meningeal infection may cause thrombophlebitis.

Brain abscess *per se* is a rather rare cause of meningitis. An unrelieved abscess almost invariably discharges into a ventricle, perhaps intermittently, and this is marked by sudden episodes of ventricular infection and meningitis.

Infection may reach the meninges by the blood stream. The route of entry in cases of clinically acute "primary" meningitis is probably by the choroid plexuses, and the more chronic meningeal infections, such as tuberculosis and syphilis, are initiated at the perivascular spaces.

Blood-stream infections

Marked vascularity of the surfaces of the brain, cord and nerve roots is apparent in acute leptomeningitis. Extensive fibrinous deposits, of a green or yellow colour, in the subarachnoid space are typical of pneumococcal and, less frequently, of staphylococcal infections. These deposits are apt to block the subarachnoid fluid pathways; this has an important bearing upon treatment with penicillin.

Morbid anatomy

(3) Clinical picture

Headache and pyrexia usher in the syndrome. The pulse rate increases. Nuchal pain, a sensation of stiffness in the neck and demonstrable rigidity of cervical muscles are very early features. The classical Kernig's sign usually appears a little later, because the majority of meningeal infections arise from infective foci within the head.

The importance and frequency of thrombophlebitis of the brain in the pathology of meningitis has been stressed above, and it should be added that some degree of superficial encephalitis coexists in most cases. Thus a wide variety

of focal cerebral dysfunctions form part of the clinical picture in different cases. The vascular disturbance of thrombosis is responsible for continuous and perhaps widespread muscle twitchings or for frank epileptic attacks, which are either focal or generalized. Aphasia, paresis of limbs or of eye movements, polyuria perhaps, and other focal derangements are quite frequent. The state of consciousness seldom remains normal for long; if sought for, evidence of some degree of confusion, disorientation, hallucinosis, or impairment of insight and memory is likely to be found almost from the outset. Later, delirium supervenes, either terminally, or earlier if the convexity of the hemisphere is predominantly affected. Affection of cranial nerve functions, especially of the motor-oculi group, is frequent in basal meningitis, and papilloedema is likely to occur if the fluid pathways at the base of the brain are obstructed by exudate.

If the infection cannot be controlled, emaciation, retraction of the head and flexion of the limbs become marked. Death is due to toxæmia or to encephalitis of the brain-stem, and rarely to high intracranial pressure.

(4) Special diagnostic methods

Examination of the fluid obtained by lumbar puncture is the most important special diagnostic procedure. (*See Brain—Neurological Investigation*, Vol. 2, p. 392.)

*Examination
of cerebro-
spinal fluid*

In acute pyogenic meningitis the cerebrospinal fluid is opalescent, turbid or frankly purulent. In subacute or indolent infections the fluid may appear to be clear but may nevertheless contain up to about 300 cells per cubic millimetre. The cell type is exclusively or predominantly polymorphonuclear at first, but the proportion of lymphocytes rises as infection is being overcome. In subacute or indolent infections, also, lymphocytes abound.

*Prevention of
coagulum in
specimens*

It is usual for a coagulum to appear in the fluid on standing; this entangles many cells and seriously vitiates the cell count. This can be circumvented by adding a few drops of sodium citrate solution to the specimen.

As a guide to treatment, it is important to have the infecting organism identified at the earliest possible moment, because the chance of identifying it rapidly disappears when treatment is under way. The organism may be seen in

Cold storage

81
cells.

The bacteriologist should always be informed if penicillin or sulphonamides have been given, so that appropriate steps may be taken to neutralize the inhibiting effect of traces of those substances upon cultures.

Biochemical estimations are of subsidiary value in acute pyogenic meningitis. As resolution is occurring, the total protein content assumes importance. A rising total protein figure should arouse suspicion of abscess in the brain or of loculation in the subdural or the subarachnoid space; obstruction of spinal fluid pathways is also a possibility. Estimation of chlorides and of sugar should also be undertaken. These are not affected in virus infections.

*Lumbar
manometry*

The lumbar fluid pressure and the response to jugular compression should be observed. In stout patients full flexion of thighs on trunk may compress the abdomen and give a falsely high reading; in this event it is necessary merely to

reduce hip flexion slightly after the needle is in place. Demonstration of free spinal fluid conduction should be performed by simultaneous compression of both jugular veins, and care should be taken to see that the patient is not holding his breath the while. Unilateral jugular compression should be reserved for demonstrating lateral sinus thrombosis (no response on affected side; full response—equivalent to bilateral compression—on patent side).

The method of puncturing the cisterna magna is given in Fig. 37.

(5) Differential diagnosis

The meningismus characteristic of subarachnoid haemorrhage simulates that of meningitis. Cases with spontaneous haemorrhages as their basis have an abrupt and perhaps dramatic onset; usually there is sudden or rapid loss of consciousness.

In the case with fractured skull and concomitant brain injury with haemorrhage, meningitis may also develop by infection through the fracture. Such a case presents difficulty until a differential cell count of the cerebrospinal fluid has been undertaken and films have been examined for organisms.

(6) Treatment

Apart from the general treatment of meningitis, such as, for instance, the control of fever and the maintenance of fluid intake, the main line of treatment is the application of antibacterial remedies; this includes taking steps to ensure that the drugs employed are enabled to reach the whole of the infected area in adequate concentration. If the intracranial pressure is raised, measures are required to restore it to normal.

(a) Penicillin therapy

Penicillin has revolutionized the management of the majority of cases of acute pyogenic meningitis which are seen in surgical practice. It should be administered intrathecally in daily doses of about 30,000 units dissolved in penicillin

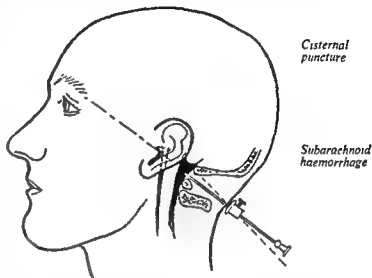


FIG. 37.—Under local anaesthesia, in the lateral position with the head slightly flexed, a lumbar-puncture needle of about 20 gauge, carrying an adjustable stop set at 5.5 centimetres from the tip, is introduced in the midline above the tip of the spine of the axis vertebra. It is directed rather above the inter-meatal line and is likely to strike the occipital bone behind the foramen magnum. The needle is then withdrawn some 3 or 4 centimetres to subcutaneous depth and is re-directed slightly lower, to pass through the occipito-atlantoid membrane which is tough and easily recognized. The stylet is withdrawn; if fluid does not flow out, the needle is inserted farther, 2 millimetres or so at a time, until fluid is obtained. The point of the needle must not be inserted more than a maximum of about 8 millimetres beyond the membrane.

The cisterna magna lies at 4.5–7 centimetres depth from the surface in normal subjects, depending upon nutrition and development.

General considerations

Intrathecally

3 millilitres of saline solution. Lumbar fluid is withdrawn as requisite for examination or to reduce pressure before injection of the penicillin. If the infection is virulent, the dose should be repeated 12-hourly during the first 2 or 3 days.

Intramuscular penicillin

In addition, regular intramuscular injections are required to maintain an adequate antibacterial level in the circulating blood and in the cerebrospinal fluid by diffusion from the blood stream. Intramuscular doses of about 30,000 units of penicillin 4-hourly are appropriate in the adult. The need for continuation of systemic administration must be gauged by clinical improvement and by restoration of the cerebrospinal fluid to normal.

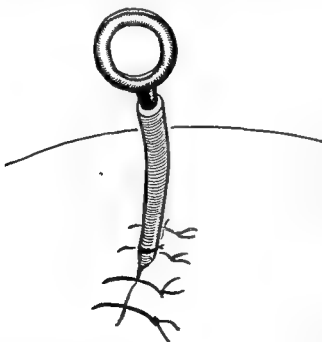
Pure penicillin

Only pure penicillin should be used for intrathecal injection. If impure preparations are employed, severe transient lumbo-sacral root pains and temporary retention of urine may occur. Moreover, instances of disastrous spinal arachnoiditis have been seen, attributable to the use of unduly irritant preparations of penicillin.

Importance of manometry

When administration by the lumbar route is in progress, lumbar manometry should be repeated at least every 2 days. Curdy, purulent exudate is apt to obstruct the fluid pathways in the spinal subarachnoid space, preventing the necessary free diffusion of penicillin in the circulating fluid. This is a feature particularly of pneumococcal and, in lesser degree, of staphylococcal meningitis. On the first hint of a sluggish response to bilateral jugular compression and release, cisternal puncture and manometry are required, and a comparison of the simultaneous lumbar and cisternal manometric responses

Cisternal penicillin



Intra-ventricular penicillin

FIG. 38.—Ventricular catheter, with wire spigot. The long ends of a scalp suture, after tying the knot, are tied round the catheter to hold it in place.

is made. If a difference between the two is apparent, the cisternal route for injection of penicillin must forthwith be adopted in addition. In these circumstances it is advisable to increase the total intrathecal dose to 40,000 or 50,000 units, of which 10,000 units should be given by lumbar injection. If the above routine procedure is adopted, ventricular administration of penicillin will seldom be required, but should be instituted if there is any doubt about the cisternal manometric response.

The procedure for intra-ventricular penicillin therapy involves the making of a burr-hole in

the skull at a suitable spot for puncture of a lateral ventricle anteriorly or posteriorly with a brain-exploring cannula, as for ventriculography.

A No. 3 rubber catheter is inserted along the track in the brain left after withdrawing the cannula, the scalp is closed with two layers of silk, and the catheter is secured in position by tying long ends of adjacent sutures around it (Fig. 38). The risk of infection along the catheter track can be minimized by applying a crushed tablet of calcium penicillin and a small quantity of sulphanilamide powder to the sutured wound where the catheter protrudes; this should be repeated daily. Obviously the protruding part of the catheter must be kept scrupulously aseptic in sterile gauze, and the dressing should be held in place by strips of Elastoplast securely adherent to the shaved and ether-cleansed scalp. In this manner a catheter may safely be left in position for as long as 5 days; if required for a longer period, as is often the case, it is necessary to re-insert the catheter elsewhere. In one case of pneumococcal meningitis, which relapsed repeatedly because of inefficient treatment, intraventricular injections of penicillin were maintained for 3 weeks by this method, and the infection was overcome.

*Indwelling
ventricular
catheter*

(b) Sulphonamide therapy

The sulphonamides are, so far, our only direct antibacterial remedy in acute infections which are due to penicillin-resistant organisms, although streptomycin or other similar preparation will shortly add to our resources.

Sulphamezathine or sulphadiazine are at present the best compounds to choose, because they appear in the cerebrospinal fluid in greater concentration than does sulphathiazole, and they are rather more polyvalent than sulphapyridine or sulphanilamide. Further, they are better tolerated by most patients.

*Choice of
sulphonamides*

Urea augments the antibacterial action of the sulphonamides (Ecker, 1945). There is no doubt that when the infection is barely being held in check by sulphonamides, the addition of urea in treatment may tip the balance in favour of the patient. Urea is given in amounts of 15 grammes 4-hourly by mouth, day and night, and is surprisingly well tolerated by many patients for many successive days. It is less unpalatable if given with syrup of orange.

*Urea with
sulphonamides*

If the intravenous route is found to be necessary, 2 per cent of urea in normal saline is efficacious; the rate of drip should be adjusted to correspond with the recommended oral intake.

In all cases of acute meningitis both penicillin and sulphonamide treatment should be instituted pending identification of the infecting organism. This procedure is also to be recommended for prophylactic treatment, as, for example, in cases of fracture of the skull in which blood or fluid leak to the exterior, or when operation is undertaken on an infected cranio-cerebral wound.

*Combined
penicillin and
sulphonamides*

Cessation of treatment as above should be determined by return of the cell and protein contents of the cerebrospinal fluid to nearly normal figures, and also by marked and sustained clinical improvement.

(c) Control of intracranial hypertension

A few cases of meningitis show substantially increased intracranial pressure, and if this is not brought below 200 millimetres by means of one or two lumbar punctures, continuous lumbar drainage will be found to be advantageous and practicable (Fig. 39). The needle can be left *in situ* for the greater part of each 24 hours, and this means that the nursing staff must contrive to attend

to pressure points on the skin without rolling the patient over. (Some patients on whom continuous lumbar drainage has been performed for reasons other than meningitis, have been thus managed efficiently for as long as 3 days without intermission.)

Continuous lumbar drainage can be combined with intrathecal penicillin treatment, the drainage tube being occluded for about 6 hours to allow

*Continuous
lumbar
drainage*

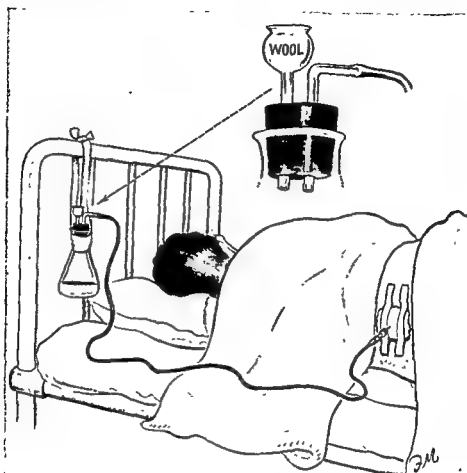


FIG. 39.—Continuous lumbar drainage. The flask can be adjusted to drain at any head of water from 300 millimetres down to zero



FIG. 40—"Record" taper mount and rubber tube for continuous lumbar drainage

diffusion. When the tube is released the overflow is set for some hours at a level of 300-350 millimetres above the lumbar needle and thus the rate of loss of penicillin in the fluid is minimized.

After it has been set up, the "Record" taper fitting (Fig. 40) on the rubber drainage tube must not be disconnected from the needle, either to test the

efficiency of the drainage system or to administer penicillin. The connexion is bound to become contaminated with blanket dust and, if interfered with, cannot be considered safe, even though it is wrapped in sterile gauze. The only permissible manoeuvre is to unfix the drainage flask and lower it to test the flow. If blocked, the lumbar needle may be rotated, withdrawn or pushed in farther, and usually this corrects matters.

Controlled ventricular drainage can be set up similarly, and it imposes much less restriction on the patient's mobility in bed (Fig. 41). If intraventricular penicillin is required simultaneously, however, the penicillin must be injected

*Continuous
ventricular
drainage*



FIG. 41.—Continuous ventricular drainage, employing Southey's rubber tubing.

through a second ventricular catheter elsewhere; this second catheter should be well protected by sterile dressings and reserved exclusively for injection of penicillin. Conservation of penicillin in the ventricles for some hours after each injection is managed as described for lumbar drainage.

5. TUBERCULOUS MENINGITIS

(1) Pathology

This serious condition occurs either as part of a miliary tuberculosis or as a "primary" tuberculous meningo-encephalitis. In either instance the infection is blood-borne from a focus or foci elsewhere. The meningo-encephalitic type arises from tubercles established in the perivascular spaces or by superficial spread from multiple and perhaps confluent cortical tubercles; in contrast to the miliary type these are usually grouped in one or several areas of the brain, and this provides an explanation for the wide variety of neurological dysfunctions which may form part of the early syndrome. "Tuberculoma" of the brain is subcortical and is often solitary. Tuberculomas are not very apt to cause meningitis; indeed they indicate good resistance to the infection, usually become calcified and ultimately even "dead" bacteriologically. Operative disturbance of a tuberculoma, short of clean and complete extirpation, can cause tuberculous meningitis; this operative complication is

certain if a cerebellar tuberculoma is cut into during removal, but it is by no means necessarily so if the lesion is in the cerebrum.

(2) Clinical features

The disease is commonest in young children, but no group up to middle age is exempt. The onset is apt to be insidious, perhaps occurring after an exanthem or other lesser ailment. Loss of appetite or of weight, and irritability, may be features of the early stages. Headache, fever and perhaps vomiting appear, and at this stage one or more focal or generalized convulsions may occur. The pulse tends to be slow. The headache is usually severe and in children causes the characteristic cry. Pyrexia is at first mild and rigidity of the neck is frequently delayed in onset.

As the disease advances, apathy or stupor supervenes, emaciation proceeds and meningismus increases. The pupils, which are often small at first, become unequal and react abnormally; motor-oculi palsies are frequent.

The intracranial pressure may be normal or increased. Specimens of cerebrospinal fluid early in the disease may appear limpid, but usually a tell-tale fine coagulum develops on standing. When shaken gently, this becomes compacted into the centre of the tube, giving the typical "spider-web" appearance. The bacillus, if present, is most likely to be found in this coagulum. (It is to be noted that a very similar spider-web effect is to be seen in some specimens from cases in which pyogenic meningitis is resolving under treatment.)

The cellular reaction in the fluid is largely lymphocytic, but occasionally at an early stage polymorphonuclear cells predominate.

Reduction of the chloride content (650 milligrams in 100 cubic centimetres, or less) is very common and characteristic, although a normal figure does not exclude early tuberculous meningitis.

(3) Treatment

Streptomycin is now on extensive clinical trial and it is to be hoped that the encouraging results obtained in some selected early cases will be extended to all types of case as knowledge in this particular field advances. Apart from such treatment, which is directed specifically against the infection, general measures for the management of any case of meningitis, and the control of intracranial tension when that is raised, are applicable.

6. SYPHILITIC MENINGITIS

This condition comes within the province of venereology, but in respect of its late effects—especially adhesive arachnoiditis and progressive hydrocephalus—it may require the attention of the surgeon (see page 81).

7. COMPLICATIONS AND SEQUELAE OF MENINGITIS

(1) Loculation abscess

Pus may accumulate in the subarachnoid or subdural space, or in both, and the term loculation abscess seems appropriate. It is synonymous with the subdural empyema described by some writers.

Localized suppuration between the brain and the dura mater has its origin fundamentally in the persistence of viable organisms in thrombosed and infected cerebral or cerebellar veins. The associated meningitis is of subsidiary

Lymphocyte
response in
cerebrospinal
fluid

Reduced
chloride
content

Pathology

importance. The cause of lingering infectivity may be either an initial or an acquired bacterial resistance to penicillin, or inadequate dosage, or mechanical impediments to free diffusion of penicillin in the subarachnoid space.

In the days before penicillin was available fewer of these cases were seen, because meningitis dominated the picture and usually carried off the patient before extensive suppuration could occur. Even when these abscesses were drained, the results were melancholy. The situation is no better today when we have to deal with penicillin-resistant infections of this type.

Loculation abscesses are often multiple and may be located near or within the median longitudinal fissure. This latter type is associated particularly with acute frontal sinusitis and spreading frontal osteitis. It may present peculiar difficulty in diagnostic localization and in treatment, as also may a purulent collection in the middle fossa in relation to the medial part of the temporal lobe. Other cases show loculations on the convexity and at times virtually the whole of this aspect of the hemisphere may be found bathed in pus if the accumulation has been in the subarachnoid space. Small multiple loculations are likely to have originated in this area. *Morbid anatomy*

The occurrence of loculation abscess in a case of acute meningitis should be suspected when clinical improvement is arrested and headache, perhaps vomiting, and focal signs of cerebral dysfunction, appear. Recurrence of pyrexia, increased or perhaps diminished pulse rate, apathy, drowsiness and incontinence are probable developments. Papilloedema and a raised fluid pressure are likely to be found. *Clinical course*

Lumbar puncture may be dangerous in suppurative intracranial conditions unless formal operative treatment can be undertaken without delay.

The cerebrospinal fluid shows some increase of cells, 20-200 per cubic millimetre perhaps, with a variable proportion of polymorphonuclear leucocytes. The total protein content is certain to be increased. *Cerebrospinal fluid*

The syndrome closely simulates that of brain abscess; indeed, having a common basis of thrombophlebitis, intracerebral and extracerebral loculi often develop concurrently.

If localization on neurological evidence is uncertain, recourse must be had to ventriculography and operation should be planned to follow in sequence thereafter. Incidentally, it should be noted that encephalography by lumbar or cisternal air-replacement is contra-indicated if the intracranial pressure is high. *Localization*

Treatment

Two or more burr-holes should be made in the skull, choosing primarily the site indicated by clinical evidence or, if necessary, by special investigations. If the site for cranial puncture has been well chosen, pus will be liberated on opening the dura mater; it may be in a locus or may extend widely over the hemisphere. The probability of multiple loculi must be kept in mind. The extent of the purulent collection can be assessed by inserting a No. 3 rubber catheter as a flexible probe in all directions within the cavity. Special attention should be paid to the region of the supero-medial border of the hemisphere if suppuration seems to have affected that area; one or more burr-holes should be made about 1 centimetre from the midline. Enlargement of such paramedian burr-holes with bone rongeurs may be necessary. *Operative treatment*
Collections near midline

to allow of opening the *dura* clear of large cerebral veins or dural venous lacunae, and to give access for evacuation of pus collected between the falx and the hemisphere.

When patients are suspected of having either multiple or extensive loculations, many burr-holes should be made. Rubber catheters of No. 3 size should be inserted and should be disposed strategically so as to allow injection of penicillin solution to each infected cavity and to all parts of the larger cavities. Several catheters may have to be inserted through a burr-hole to achieve this purpose. The catheters should be brought out through small independent stab wounds close to the incision, and the incision should be closed with two layers of silk, notwithstanding the existence of infection. The margin of the burr-hole may require trimming here and there, if nipping of any catheter seems probable.

It is fortunate that in most cases of subdural abscess the organism is penicillin-sensitive. When penicillin is not applicable the prognosis—even with sulphonamides—is very grave if the loculations are multiple or extensive. Dosage of penicillin depends upon the number and extent of the cavities to be treated; a total of 30,000–50,000 units of pure penicillin dissolved in 3–5 millilitres of saline solution should be injected at each session, and should at first be given twice, or in bad cases, thrice, at regular intervals throughout the 24 hours.

Intramuscular penicillin is also required in all cases. Systemic administration alone has been found to be ineffective in brain abscess and may be presumed similarly so in subdural abscess. This obtains only so long as the tension within the abscess cavity is high. Thus, the pus obtained at the first tapping of a brain abscess shows a very low antibacterial titre notwithstanding full systemic penicillinization for the preceding 24 or 36 hours, but almost at once the titre rises to, and remains at, a high figure when the tension in the abscess is lowered (Sedzimir, 1945).

The prognosis is always potentially serious, even in cases in which penicillin can be employed, because of the difficulty, in some cases, of locating and

(2) Arachnoidal adhesions

After an attack of meningitis, the arachnoid membrane is apt to be thickened and adherent to the pia mater, so that the subarachnoid space is in parts obliterated. These adhesions may obstruct the circulation of cerebrospinal fluid, particularly if they occur at the anatomical “bottle-necks”—at the foramen magnum, at the incisura tentorii or at the basal cisterns. The subarachnoid spaces on the surfaces of the hemispheres become distended with fluid if the adhesions are so located as to interfere with resorption of cerebrospinal fluid at the Pacchionian villi near the supero-medial border of the cerebrum. This condition is termed chronic external hydrocephalus (the alternative name, chronic serous meningitis, is misleading because the fluid is not “serous” and usually contains no excess of cells). Incidentally, it may be noted that the damage of acute external hydrocephalus is a resorption of cere-

Systemic
penicillin

Prognosis

Morbid
anatomy

Lastly, spinal arachnoiditis can cause compression of the spinal cord or of the nerve roots, by the formation of one or several cystic spaces containing clear, perhaps pale yellow, fluid of fairly high protein content. This fluid is probably derived from the perivascular spaces. *Spinal arachnoiditis*

Adhesions as described can follow any variety of meningitis, but it is worth mentioning that a few infections show some predilection for particular regions. Adhesions restricted to the region of the cerebellar tonsils and other structures near the foramen magnum should suggest the possibility of a syphilitic origin even in cases in which the Wassermann reaction has been found to be negative; quite often after operative disturbance of the adhesions the reaction becomes positive for a time, if the disease has a syphilitic basis. Adhesions in the posterior fossa or in the basal cisterns occur after meningococcal infections, but are not exclusively characteristic thereof. Transient chronic or very subacute external hydrocephalus occurs in some cases of otitis media ("otitic hydrocephalus") (Symonds, 1931, 1937).

Clinically, cases with arachnoidal adhesions which are causing symptoms present the general picture of intracranial or of spinal tumours. There are, usually, no clear localizing features when the adhesions are intracranial and ventriculography is required. Even when spinal adhesions are present, localization may not be well defined and contrast myelography therefore becomes necessary. *Clinical syndromes*

Diagnosis and treatment in the conditions under review call for considerable specialized experience in neurosurgery, and only an outline of operative treatment will be given here. Cerebellar decompression with removal of the posterior arch of the atlas gives the necessary access to adhesions at the level of the foramen magnum. The rather active stream of fluid from the fourth ventricle seems to be effective in preventing re-formation of the adhesions, and the results in this type of case are usually very satisfactory.

Obstructions at levels up to the incisura tentorii can be relieved by making an aperture in the anterior wall of the third ventricle above the chiasma, whereby ventricular fluid is short-circuited directly into the basal cisterns and so to the surface of the hemispheres along normal channels.

The treatment of chronic external hydrocephalus is bilateral subtemporal decompression. Mild grades of this condition respond to lumbar drainage or to repeated lumbar puncture.

REFERENCES

- Ecker, A. D. (1945) *Lancet*, 1, 176.
 Sedzimir, B. (1945) Communication to Society of British Neurological Surgeons
 Symonds, C. P. (1931). *Brain*, 54, 55.
 — (1937). *Ibid*, 60, 531.
 [References to other titles are given under Meninges—Meningitis, Acute and Chronic; Infection of Cerebrospinal Space, in the Index Volume. The subject is also dealt with under the heading of Meningitis in the *British Encyclopaedia of Medical Practice* (1938), Vol. 8, p. 495.]

MOUTH AND PHARYNX, MALIGNANT DISEASE OF

BY SIR STANFORD CADE, K.B.E., C.B., F.R.C.S., M.R.C.P.
SURGEON, WESTMINSTER HOSPITAL, LONDON; CONSULTING SURGEON, MOUNT
VERNON HOSPITAL AND RADIUM INSTITUTE, NORTHWOOD, MIDDLESEX.
CONSULTANT IN SURGERY TO THE ROYAL AIR FORCE

AND

E. STANLEY LEE, M.S., F.R.C.S.

SURGEON, OUT-PATIENT DEPARTMENT, WESTMINSTER HOSPITAL, LONDON.
SURGEON, MOUNT VERNON HOSPITAL AND RADIUM INSTITUTE, NORTHWOOD,
MIDDLESEX; SURGEON, RADIUM CENTRE, GUILDFORD, SURREY

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INTRODUCTORY DEFINITION

In the following article, the term "mouth and pharynx" is held to include these sites: lips, tongue, mouth floor, gums, cheek, hard and soft palate, fauces and tonsil, maxillary antrum, nasopharynx, oropharynx and laryngopharynx. Inasmuch as the great majority of malignant tumours in these situations are squamous-cell carcinomas, this type alone will be considered in detail. Other malignant tumours will be mentioned in passing. Tumours of the jaws are not considered.

PART I

NATURAL HISTORY AND DIAGNOSIS

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1. AETIOLOGY

(1) Age and sex incidence

234.] In the region of the mouth and pharynx striking and puzzling differences in sex and age incidence are encountered between otherwise similar tumours occurring in different sites. Thus, for the lip, the anterior two-thirds of the tongue, and the buccal mucosa, there is in most countries an 80-90 per cent preponderance of male cases over female; the exception is Scandinavia, where the incidence in women is 40 per cent. For the soft palate this applies equally, but in sharp contrast the hard palate is affected equally in the two sexes, as also is the posterior third of the tongue. In the pharynx are found two limited zones in which women develop growths far more commonly than men: they are the back of the oropharynx and the post-cricoid region. In this latter situation, moreover, the disease commonly arises 10-15 years earlier than in other neighbouring sites. The piriform fossa and the aryepiglottic folds are affected in males predominantly.

(2) Known carcinogenic factors

Such factors as are known to have a carcinogenic importance may be divided into two main groups: the local irritants and the deficiency states.

(a) Irritants

Among these may be mentioned heat, chewing of tobacco and betel nut, alcohol, and the trauma and gross sepsis due to bad teeth. This last factor probably accounts for the preponderance of tongue



FIG 42—Extensive leucoplakia of tongue, palate and cheek, showing development of carcinomatous change.

Leucoplakia

cancer in the poorer classes, whose teeth are in general less well tended. Special mention must be made of syphilis, though this is of diminishing importance in England. The various forms of leucoplakia, especially the associated cracks and warty formations, must be recognized as pre-cancerous and treated accordingly (Fig. 42). Now, however, the gross leucoplakia of former days is seldom seen; small patches of thin, sore, red



FIG. 43.—Minor degree of leucoplakia of the tongue consisting of smooth tender areas with a few flecks of white heaped-up epithelium. The lesion, which has been observed for several years, is almost stationary. The Wassermann reaction is negative.

Plummer-Vinson syndrome

epithelium, or smooth, scarred-looking areas on the tongue, lip or buccal mucosa are all that are now commonly seen, and there is often no recognizable syphilitic basis (Fig. 43).

These irritants are found to operate chiefly in the anterior part of the mouth. Their influence in the posterior part of the tongue and in the pharynx is questionable.

(b) Deficiency states

The atrophic condition of the mucosa known as the Plummer-Vinson syn-

drome is generally considered to be a deficiency state. It appears to underlie a large proportion of cases of oral and pharyngeal cancer in women, and its early recognition and treatment are therefore most important. In this syndrome a thin, red, inelastic mucosa, with a small buccal orifice having a tendency to crack at the angles (cheilosis) and a smooth red tongue, are associated with some or all of the following: an iron deficiency anaemia (sideropenia), dysphagia (often of long standing), moderate splenomegaly, and spoon-shaped finger-nails (koilonychia). This state occurs almost exclusively in women.

Vitamin deficiencies

Recently evidence has accumulated that deficiencies of some of the vitamin B complex (riboflavine and perhaps also nicotinamide and pyridoxine) give rise to degenerative changes with atrophy of the mucous membrane and submucosal fibrosis, changes which are pre-cancerous. This may explain the tendency for chronic alcoholics to develop buccal cancer, and the poor results often obtained in treating them: for in chronic alcoholism the alimentary absorption of these vitamins is impaired, and at the same time the metabolism of alcohol uses up the available supplies.

(c) Other factors

Bowen's disease

Other pre-cancerous conditions of unknown origin are Bowen's disease, a chronic superficial ulceration of the mucosa (as also of the skin) perhaps allied to Paget's disease of the nipple and occasionally of the penis or other sites, showing lymphocytic infiltration and downgrowth of epithelium, and tending eventually to become frankly malignant.

This type of neoplasm extends widely on the surface, and often is multicentric in origin. It is slow in spread, but eventually gives rise to lymph-gland metastases. Nothing is known of its causation, and some pathologists consider it to be from the commencement a carcinoma of low-grade malignancy. Lichen planus occasionally occurs in the mouth as flat, milky-white patches or as an irregular "lace-work" of fine white dots or streaks, usually associated with simultaneous skin lesions, and this also is believed by some authorities to be pre-cancerous.

2. PATHOLOGY AND NATURAL HISTORY

(1) Macroscopic types of carcinoma

In gross structure, a carcinoma in any of the regions now under consideration usually conforms to one of three well-known types: the nodule, the ulcer or fissure, and the warty or papilliferous growth. Examples of these types as they occur in all the sites are depicted in the accompanying figures. Such variations as occur will now be described under the anatomical headings.

(2) Carcinoma of the lips

This disease is much commoner on the lower than on the upper lip;



*Lichen
planus*

(a)



*The nodule,
the ulcer and
the wart*

(b)

FIG. 44.—Two early carcinomas of lip. (a) Shows very shallow ulceration which has been covered with a small crust (b) Shows ulceration, still shallow, but apparently of multicentric origin.

Site

carcinoma of the upper lip is rare, particularly in males, and is of graver prognosis than are lesions on the lower lip. Similarly, carcinoma at the angles of the mouth has a worse prognosis than that affecting the straight

part of the lower lip, which is one of the slowest cancers to metastasize.

Early appearances

FIG 45.—Massive fungating carcinoma of the lower lip.

The growth may begin as a harmless-looking crusting or wartiness of the lip, the heaped-up epithelium being shed to produce shallow ulceration (Fig. 44). In other cases a small crack or fissure may arouse suspicion by marginal induration. The lesion may correspond accurately to the site at which a pipe is habitually carried, or to the point of contact of a jagged or decayed tooth.

In the more advanced stages the lesion usually presents as an infected ulcer with a more or less heaped-up and exuberant edge. Occasionally, however, a massive proliferative lesion is seen (Fig. 45).

Lymph drainage

It may reach a large size before enlargement of the submental, the submandibular or the deep cervical glands becomes evident. Lymph may drain direct to any of the above-named groups, so that a complete examination of the neck is essential in every case. Fig. 46 shows a carcinoma of the upper lip, with bilateral involvement of the submandibular lymph glands.

Microscopically, the growths are squamous-cell carcinomas, with or without cell nests. Sometimes a relatively undifferentiated histological picture is met with.

(3) Carcinoma of the tongue

It is usual to distinguish between growths of the anterior two-thirds of the



FIG. 46 —Carcinoma of the upper lip, showing bilateral involvement of the submandibular lymph glands.

Histology

tongue and those of the posterior third, which differ in their natural history and present quite a different problem for treatment. Cancer in the anterior part of the tongue commences either as a small hard node, warty or button-

*Anterior
two-thirds
of tongue*

like, a crack or fissure, or an ulcer (Fig. 47). In addition, there is seen an infiltrative type, spreading widely in the substance of the tongue. The organ becomes more and more fixed, with relatively little surface evidence of disease. Sometimes in this type there are multiple outcroppings of the underlying disease at widely separated parts of the tongue.



*Clinical
types*

(a)



Symptoms

*Later
evolution*

(b)

FIG 47—Carcinoma of the tongue. (a) ulcerating type, (b) nodular type.

Not infrequently, the tongue may be seen to be affected with leucoplakia, in which the carcinoma has developed in a crack or a warty patch. In the early stages carcinoma is painless, though when the floor of the mouth or the alveolus becomes involved by extension pain may become severe; referred pain to the ear is frequent and is characteristic of extension to the floor of the mouth. Gross infection also increases pain. In the advanced stages the clinical picture is that of great misery, with unceasing salivation, great difficulty in speech, mastication and swallowing, constant pain, and trismus. Death may occur from inhalation pneumonia, from haemorrhage due to invasion of the lingual vessels, or less mercifully from cachexia resulting from starvation, pain, loss of sleep, and toxæmia. Visceral metastases occur late, but may become manifest if life is prolonged by arrest of the primary disease; skeletal metastases also occur. The first lymph glands to become enlarged may be the submental, the submandibular, or the upper deep cervical. Unless the growth is laterally situated the glandular spread is quite likely to be bilateral, as there is no barrier to the spread of lymph across the midline.

*Posterior third
of tongue*

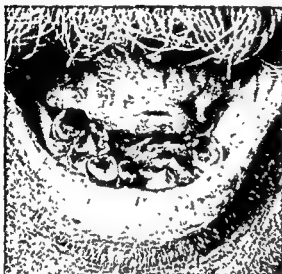
*Lympho-
epithelioma*

The posterior part of the tongue is remarkable for the bulky, undifferentiated carcinomas which occur there. These include the histological types known as the "transitional-cell carcinoma" and "lympho-epithelioma", noted for their rapid growth and early involvement of lymph glands, and their ready response to irradiation. This is, however, only too often followed by speedy and massive recurrence, so that the ultimate prognosis is poor. Lympho-epithelioma is now considered by some authorities, especially the Swedish school, as mesodermal and not epithelial in origin—that is, as a lymphosarcoma.

Ulcerative and infiltrating carcinomas also occur.

(4) Carcinoma of the mouth floor, alveolus and cheek

These sites may be considered together, because the carcinomas exhibit much the same characters, and moreover often involve—except in the earliest stages—more than one of the sites. The ulcer is the commonest form on the mouth floor. At first circular or oval, it often comes to present a folded appearance, being partly



(a)



(b)



(c)

FIG. 48.—Carcinoma of floor of mouth (a) The grossly infected teeth are shown, with the "folded" malignant ulcer behind them. Such teeth should be extracted at the commencement of treatment. (b) and (c) Extensive carcinoma of the lower gum and floor of mouth, with involvement of the jaw.

*Mouth
floor*

on the mouth floor and partly on the under-surface of the tongue (Fig. 48). On the cheek the warty and plaque-like lesion is also common, and very often accompanying leucoplakic patches may be seen. On the alveolus, the tumour usually appears as a nodular, warty or fungating mass which, if teeth are present, may resemble the granulomatous epulis sometimes seen in relation to caries.

Exact observation of the extent of these mouth tumours is important, for not only the prognosis but the choice of treatment depends largely upon it. In the later stages of all these growths the cheek or mouth floor is penetrated, with involvement of the skin and formation of salivary fistulae. Enlargement of lymph glands occurs much earlier than with carcinoma of the lip, but the prognosis in early cases, without glandular spread, is by no means bad. Five-year "cure" rates of 50-60 per cent for such cases are claimed.

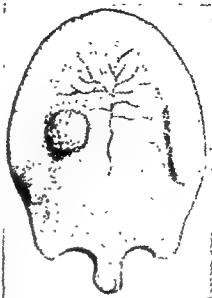


FIG. 49—The hard palate, showing carcinoma of the warty type.



FIG. 50—Massive swelling of the palate, with central ulceration, resulting from invasion by a tumour of the maxillary antrum

rounded submucosal tumours, ^{gland tumours} they eventually ulcerate or fungate. Lymphatic dissemination tends to be later with these tumours.

(5) Carcinoma of the hard palate

As has already been stated, growths of the hard palate differ considerably from those of the soft palate. They occur as warty, often circular lesions (Fig. 49), or sometimes as shallow ulcers with raised edges.

Penetration of the bone is not usually early, and a considerable spread over the surface of the palate before this happens is the rule. It is important to distinguish primary carcinoma of the hard palate from secondary involvement of the part ^{Confusion with antral tumours}

by penetration of antral or nasal growths (Fig. 50), and this point is considered more fully in the section on Diagnosis.

(6) Carcinoma of the soft palate, fauces, tonsil and back of tongue

These are much less favourable sites for carcinoma than those so far considered. This is due partly to the technical difficulties of treatment, but also to the undifferentiated types of tumour met with, showing rapid growth and early lymph-gland involvement. The macroscopic appearances are similar to those in the other sites, except that a bulky, fungating type is more commonly

seen, sometimes reaching quite considerable dimensions. Thus a carcinoma of the tonsil may form a projection bulging across the midline and almost meeting the normal tonsil of the other side. Faucial carcinomas, and those of the soft palate, often form extensive ulcerating lesions (Fig. 51), with a variable but often gross degree of secondary infection producing a great deal of pain and dysphagia. Such lesions spread early from their origin in the fauces to the tongue, the alveolar mucosa of both the upper and the lower jaw, and the lateral wall of the oropharynx. Bilateral lymphatic metastases are quite com-



FIG. 51.—The soft palate, showing ulcerating carcinoma.

mon. In the later stages trismus due to involvement of the masticatory muscles, penetration of the soft palate with perhaps pathological communication between nose and mouth, and marked salivation with blood-staining are seen. Profuse haemorrhage may be a terminal event.

(7) Carcinoma of the maxillary antrum

About two-thirds of all antral tumours are carcinomas, most of them being squamous-cell; basal-cell, columnar-cell, and adenocarcinomatous growths also occur. The remaining one-third consists of sarcomas of various kinds, from slow-growing fibro-sarcomas and giant-cell tumours to round-cell sarcomas of the most malignant character, and includes a smaller group of rarer tumours such as myxosarcoma and plasmocytoma. The rate of development and the clinical picture vary greatly according to the line of spread of the tumour, but the manifestations are much the same whatever the histological type.

At first, the tumour is confined inside the bony box of the antrum, related to the orbit above, the cheek laterally, the nose medially, the palate below and the pterygo-maxillary fossa behind. Blockage of the opening by which the antrum drains into the middle meatus of the nose may lead to fullness and discomfort, and superadded infection may produce actual pain. This pain may in some cases lead to dental investigations and treatment, and Fig. 52

Graver
prognosis

Histology

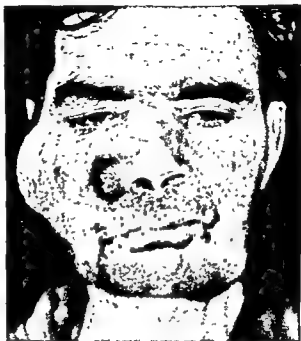
Relations of
the antrum

Early
symptoms

illustrates what may happen to a patient while the teeth supposedly responsible for his symptoms are being dealt with.

At this stage, too, the infection may produce a crop of nasal polypi, with unilateral nasal obstruction. Rapid recurrence of the polypi after removal should be looked upon with suspicion.

Sooner or later, the antral walls will be transgressed, and the subsequent picture depends upon the direction of this spread. If the growth extends through the outer wall, swelling of the face is produced (Fig. 52); if the orbital floor is involved, proptosis and double vision result. If the hard palate is invaded, at first a bulge, then a malignant ulcer results which may resemble a primary cancer of the palate itself (Fig. 50). In other cases the outer nasal wall may be destroyed, leading to unilateral nasal obstruction with offensive and at times blood-stained discharge. Glandular involvement is not as a rule early, but about one-third of the cases have palpable glands when first seen.



Late evolution

FIG. 52.—Advanced carcinoma of the maxillary antrum, with invasion of the cheek, nose and palate and bilateral lymph-gland involvement which developed while the patient was undergoing treatment for supposed dental sepsis.

Lymph-gland involvement

(8) Carcinoma of the nasopharynx

Tumours of the nasopharynx may be squamous-celled, and are often of the anaplastic varieties including the "transitional-cell" and "lympho-epithelioma" types—radio-sensitive tumours of rapid growth and high malignancy. The histological picture is often obscure and the distinction between sarcoma, carcinoma, and endothelioma often hazy. The other rare tumour found in young patients is the fibroma or fibro-sarcoma.

The nasopharynx is related above to the basis cranii, and laterally to the Eustachian tubes and the palatal and pterygoid musculature. The clinical effects of a nasopharyngeal tumour will depend upon the bulk of the mass, and upon its invasive powers. If large, the tumour will cause nasal blockage, post-nasal catarrh or bleeding, and a sense of fullness or discomfort. The Eustachian orifice may become obstructed, resulting in deafness. The invasive properties of nasopharyngeal tumours are sometimes remarkable, and pain and trismus may be produced by direct spread into the pterygoid region involving the masticatory muscles and compressing branches of the fifth

Symptoms

*Cranial
nerve palsies*

*Lymph-gland
involvement*

nerve. The basis cranii may be penetrated, after which the tumour characteristically involves the cranial nerves, particularly the sixth and the third, sometimes the fifth and seventh, and less commonly the eighth and the fourth. Cases are encountered in which these extensive cranial nerve palsies are seen, and there is deep and severe pain, yet the nasopharynx remains free from any demonstrable tumour mass, owing to the small size and predominantly invasive character of the latter. Involvement of the cervical lymph glands may be early or late, and is quite commonly bilateral. Occasionally, the cervical mass may be the presenting symptom.

(9) Carcinoma of the oropharynx and laryngopharynx

Here the familiar macroscopic forms are encountered again—the nodular, the warty and the ulcerating cancers. Microscopically, they are all squamous-cell carcinomas, varying from the well-differentiated types to the anaplastic type. But it is useful to distinguish four main sub-groups of tumours, widely divergent in their age and sex distribution.

(a) Oropharyngeal tumours

These occur mainly on the posterior wall but sometimes on the lateral wall of the gullet. In the former situation, the patients are mostly female, and in both locations the disease is one of elderly people. These tumours may reach a considerable size in the relatively spacious oropharynx without producing serious symptoms. At most the patient notices a little discomfort in the throat, with some salivation and perhaps slight blood-streaking of the expectorated mucus. Later, the sputum may become foetid, owing to infection of the growth, and dysphagia develops. Lymph-gland involvement is not, as a rule, early.

(b) Epilaryngeal tumours

These growths mostly arise on the aryepiglottic folds, which are well illustrated in Fig. 53. As the disease spreads back along the fold to the arytenoid



FIG 53.—The tongue and pharynx viewed from behind. The larynx has been opened posteriorly and the piriform fossae are displayed, the right containing a penetrating carcinoma. The specimen also shows the aryepiglottic folds and the post-cricoid region.

cartilage and forward to the epiglottis, alteration of the voice becomes noticeable. Later this progresses to hoarseness, and there will be dysphagia.

*Early
alteration
of voice*

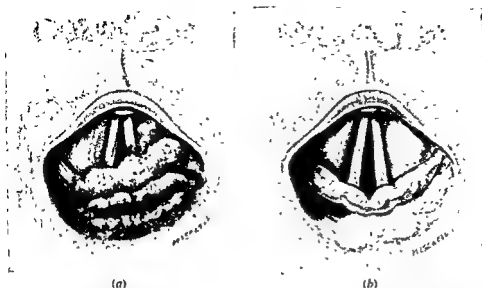


FIG 54.—Post-epicoid carcinoma: (a) Ulcerating lesion before treatment, (b) Same case 1 month later, showing radium filming and disappearance of the growth.

with expectoration of blood-stained muco-pus. There may be a complaint of pain in the throat, especially in ulcerated lesions. Because of the early alteration of the voice, growths in this group are, on the average, seen earlier than the others.

(c) *Carcinoma of the piriform fossa* (Fig. 53)

This pear-shaped gutter, situated between the aryepiglottic fold and the ala of the thyroid cartilage, is illustrated in Fig. 53 which shows an ulcerated carcinoma in the usual position.

Sometimes tumours arise more laterally, spreading secondarily into the fossa. A remarkable feature of this group, in contrast



FIG 55.—Lateral skiagram of air passages, showing the forward displacement of the larynx and trachea produced by a post-epicoid carcinoma.

with the last, is the clinical silence of the early stages. At the most a minor *Silent early stages* discomfort which is increased on swallowing may be noticed by the patient,

with expectoration of a little mucus. Cervical lymph-gland enlargement is often the presenting symptom.

(d) *Retro-laryngeal tumours*

These are of two kinds, the post-cricoid carcinoma occurring almost exclusively in young women (Fig. 54), and a carcinoma of the posterior wall of the laryngopharynx which is found more commonly in males than in females. Symptoms are due to the obstruction of the upper aperture of the oesophagus, and to infiltration of the arytenoid cartilages and muscles. The tumours are usually out of sight of the laryngeal mirror, because only the upper edge reaches to the top of the arytenoids, but the presence of the underlying tumour may be suspected from the oedema of these structures and the characteristic pool of mucus that collects behind the larynx. The tumour may extend downwards for a considerable distance, producing a mass which displaces the larynx forwards and may therefore be visualised in a lateral skiagram (Fig. 55). Lymph-gland enlargement is often already present when the patient is first seen.

Laryngoscopy

3. DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

(1) *Technique of clinical examination*

(a) *Inspection*

The patient should first cleanse his mouth and throat by gargling, to remove mucus and food debris, and any particles left clinging to the surface of a lesion should be sponged away with pledgets of damp cotton-wool held in a forceps such as Tilley's. The lips and the tongue should always be gently mopped with gauze in order that the surface may be examined dry. Minor changes such as those of early leucoplakia are thus rendered more obvious.

Equipment

The degree of mobility of the tongue should be noted: the healthy tongue can usually be made to protrude half-way to the point of the chin. A cheek retractor should be at hand in order to display the sulci and the mouth floor clearly. For the inspection of the interior of the buccal cavity a head-lamp or mirror is essential, and the brighter the illumination the less the likelihood of overlooking small lesions. For the nasopharynx the small, angled post-nasal mirror, and for the back of tongue and larynx the larger mirror, are required. Resort should be had to a cocaine spray (10 per cent solution) if the patient is intolerant or if trismus is present, or alternatively a lozenge of amethocaine

may be sucked a few minutes before examination. Occasionally a general anaesthetic is necessary. In some cases a better view of the nasopharynx may be obtained with the electric pharyngoscope. A nasal speculum should be avail-



FIG 56—Antral transilluminator, with non-heating "Coldlite" cover. (Vann Bros)

able, and in certain cases the nasal linings may be shrunk by spraying or nacking with 10 per cent cocaine solution. For transillumination in suspension, the patient is placed in a supine position and the examination is carried out with the aid of a specially made transilluminator (Fig. 56) as required. The capacity of the

Trans-illumination

maxillary antrum may be due to an empyema or to a thickened lining from chronic antral infection, or to a tumour.

(b) Palpation

The true extent of some lesions, notably those of the back of the tongue and the base of the faucial pillars, can be better gauged with the palpating finger than by inspection. Digital examination of this region should, therefore, never be omitted. Bidigital palpation of lesions of the cheek or the mouth floor is sometimes particularly helpful.

Palpation of the neck (Fig. 57).—The patient's collar and tie must be removed. To search for lymph glands, the surgeon should stand behind the

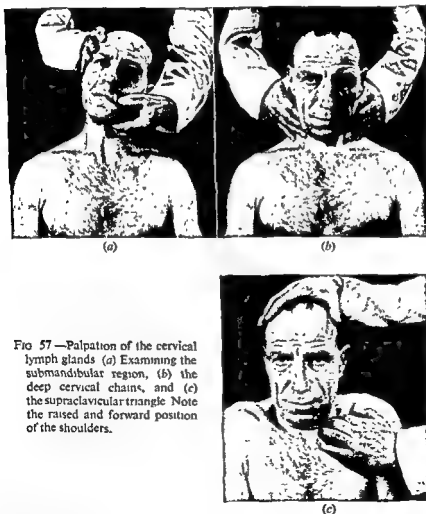


FIG 57.—Palpation of the cervical lymph glands (a) Examining the submandibular region, (b) the deep cervical chains, and (c) the supraclavicular triangle. Note the raised and forward position of the shoulders.

seated patient, and his two hands should palpate the two sides of the neck simultaneously and systematically. The search should begin with the submental region, proceed to the submandibular, then to the deep cervical, and finally to the supraclavicular groups. At each step, the patient should be instructed how to relax the structures under examination. For the submental

Submental and submandibular glands

and submandibular regions, the chin should be lowered, and if necessary the head leaned over to the side under suspicion. Further search of the submandibular regions is made with the pulps of the examining fingers turned outwards and the finger-tips hooked under the ramus of the jaw. The tissues are relaxed as completely as possible, and the finger-tips drawn downwards in contact with the jaw, much in the same way as in searching the walls of the axilla (Fig. 57 (a)). The deep cervical groups should be examined with the patient's head held forward so that the sternomastoid muscles are relaxed, and again a sideways leaning of the head may help (Fig. 57 (b)). The supraclavicular glands may best be felt if the patient shrugs the shoulders slightly and brings them forward so as to elevate the clavicles and loosen the skin covering the fossae (Fig. 57 (c)). Attention should be given to palpating the larynx, noting its breadth, symmetry and mobility.

Deep cervical and supraclavicular glands

(2) Special methods of examination

Microscopy of exudates

Radiography

In addition to those methods already mentioned, reference should be made to dark-ground microscopy of exudate from ulcers for spirochaetes, and to certain special radiographic examinations. Much information may sometimes be obtained from lateral views of the mouth, pharynx and larynx, especially if the patient distends them with air by closing the lips and nostrils and making an expiratory effort. In this way soft-tissue swellings or irregularities may be visualized on skiagrams. The best skiagrams of the lower jaw are obtained by using intra-oral dental films. The maxillary antra should be x-rayed antero-posteriorly, laterally and vertically. Stereoscopic pictures are always useful, and tomograms are sometimes of value. Finally, the advisability of a chest picture should not be overlooked. Examination for a suspected early antral tumour should, if necessary, include antral puncture so that the irrigated contents may be inspected, and perhaps Lipiodol instilled for further skiagrams. Operative exploration after the method of Caldwell-Luc is sometimes justified.

Antral puncture

(3) Biopsy: its importance and its limitations

In the diagnosis of malignancy, especially of the suspicious incipient lesion, there is only one safe rule: *when the signs do not suffice for a firm clinical diagnosis, immediate biopsy must be urged*. There is no excuse for expectant treatment, such as the trial of antiseptics or anti-syphilitic remedies. Very small lesions should be excised *in toto*, preferably by diathermy.

If a margin of healthy tissue about equal to the diameter of the lesion is included, the procedure is at the same time good therapy. When the lesion is larger, a portion of the edge should be excised, including the adjacent "normal" tissue.

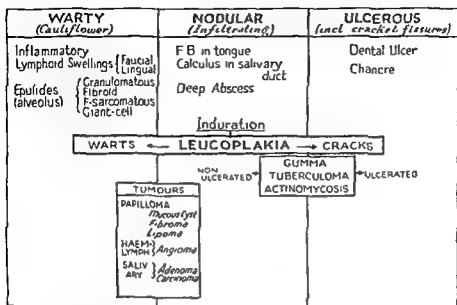
Misleading histological reports

A word of caution on the negative histological report is necessary. Since for a lesion that can be seen and felt there must be a cause, a report which reads "no evidence of carcinoma or syphilis", but offers no positive alternative, is useless and must be discarded. Either the biopsy must be repeated or the case treated on its merits. To reassure the patient and dismiss him is to invite the direst disaster.

Some of the differential points for each anatomical site in turn will now be considered. Table I sets out most of the alternatives according to the

TABLE I

Differential diagnosis of carcinoma of the tongue and mouth according to the three main clinical types of growth.



macroscopic type of the lesion, and some benign tumours of the tongue are illustrated in Figs. 58 and 59.

(4) The lips

One should always hesitate to diagnose warty lesions of the lip, especially the lower lip, as benign. The same applies to chronic fissures, especially if indurated. Occasionally a chancre may lead to confusion, though the widespread painless enlargement of the cervical glands is diagnostic. Diagnosis is made by the demonstration of spirochaetes, and of course a positive Wassermann reaction must never be accepted as evidence against carcinoma.

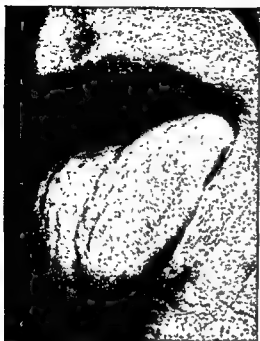
(5) The tongue

Pre-malignant lesions have already been referred to, and require the closest clinical supervision. Leucoplakia may remain stationary for months or even years; but there must be no lessening of vigilance, for the lesion may enter a progressive phase at any time and demand biopsy or more drastic measures. *Management of leucoplakia*
 Ulcers thought to be traumatic may be biopsied when the offending tooth is removed; in any event the progress of the case must be followed until the ulcer is completely healed. Inasmuch as many antiseptics have an irritant action, their local application as paints or mouth-washes is to be avoided; cleansing is best limited to the use of saline or bicarbonate solution, or Glycothymoline. *Dental ulcers*

(6) The alveolus, cheek and mouth floor

On the gum, the carcinomatous proliferation has to be distinguished from the other types of epulis, as already mentioned. Often this is only possible histologically. On the mouth floor, induration around a calculus in the sub-mandibular duct may cause confusion. Adenomas and adenocarcinomas of mucous-gland and salivary-gland origin are occasionally met with in this

situation, and are at first submucosal nodules. They are more elastic than carcinomas, and at first seem well-localized tumours. Later they ulcerate and infiltrate.



(a)

(7) *The hard palate*

The only difficulty of diagnosis is as a rule from antral or nasal growths involving the palate by direct spread. If the centre of the lesion is gently probed with a hypodermic needle, the integrity or otherwise of the bone will usually make the diagnosis clear. Examination of the nose and transillumination of the antra, as well as radiological examination, should be carried out.

(8) *The soft palate, fauces and tonsil*

These lesions are commonly ulcerated and infected, and may sometimes resemble primary, secondary or tertiary syphilitic lesions. In the first two instances, lymph-gland enlargement may arouse suspicion of syphilis.



(b)



(c)

FIG 58 — Some benign tumours of the tongue. (a) Fibroma (b) Angioma (c) Papilloma of sessile type.



FIG. 59.—Warty papilloma.

(9) The pharynx

(a) Nasopharynx

The early diagnosis of these tumours is often a matter of great difficulty. Painsstaking and repeated examination is sometimes necessary, using all the ancillary aids already described. Biopsy in this situation is especially fallacious, not only because of technical difficulties in securing exactly the right piece of tissue, but also because the histological picture is liable to be difficult of interpretation. In some infiltrating tumours, with nerve palsies and pain, the response to x-ray therapy may furnish the only absolute confirmation of the diagnosis. *Difficulty of diagnosis*

(b) Oropharynx

These tumours can usually be seen on careful inspection of the back of the throat. The difficulty is that the symptoms are so slight that the patient does not seek advice until the disease is far advanced. Once found, the lesion is usually characteristic enough to make diagnosis easy. The vertical extent of the lesion should be carefully ascertained, as extension above the level of the soft palate or below the hyoid is easily overlooked.

(c) Laryngopharynx

Some of these lesions, on the other hand, are very difficult to see. It is important to keep in mind that the commonest cause of persistent alteration in the voice, throat discomfort or expectoration of mucus is carcinoma. Success in diagnosis depends largely on an alert awareness of this fact. In the words of Wilfred Trotter (1926): "In middle-aged and elderly men any kind of abnormal sensation persistently felt in the same part of the throat should be regarded seriously. The sensation may be a tickling; it may be that of a crumb or larger body of food lodged in the throat; it may be a discomfort in swallowing saliva alone while there is no difficulty at meals. There is usually no pain in these early stages, no trouble in swallowing food, no alteration of the voice, except perhaps to a very finely discriminating ear, and no affection of the general health".

On direct laryngoscopy, the tumour may be entirely hidden behind the arytenoids; but its presence may be suspected by the oedema of the latter,

their diminished movement, and by the pool of mucus lying behind them. Forward displacement or broadening of the larynx may sometimes be evident on palpation and Fig. 55 shows the characteristic x-ray appearance due to the soft-tissue mass between the vertebrae and the larynx.

PART II TREATMENT

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1. GENERAL INTRODUCTION

(1) The alternatives available

The main alternatives available for the treatment of any cancer are surgery, radiotherapy and various combinations of these. The term radiotherapy

includes: (1) interstitial therapy with radium or radon; (2) surface therapy

sites, the degree of surgical radicalness so successful in dealing, for example,

but the intervening muscular tissues of the mouth floor remain to harbour malignant cells, which sooner or later show themselves as local recurrences. Least inaccessible of the mouth sites is the lip, but even here the limitations of

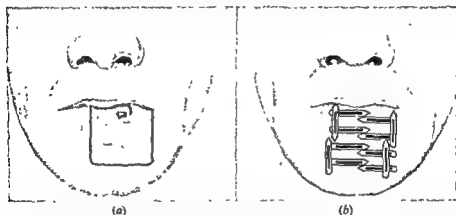


FIG. 60—Treatment of carcinoma of the lip (a) Surgery: the drawing illustrates the extent of the necessary excision (b) Radium needling: the same area is much more readily dealt with by a single-plane implant.

surgery are seen (Fig. 60) in the disfigurement which must result from really adequate excision, and which may necessitate elaborate plastic repair if a tolerable cosmetic and functional result is to be achieved.

(2) The advantages of radiotherapy

Radiation, though it has its own important limitations, can be applied to as great an area or a volume of tissue as is desirable. For this reason, and because the results at least equal those of surgery with smaller risk and less mutilation, radiation is, with certain well-defined exceptions, the treatment of choice. In the view of the authors—and it is a view shared by many others—radium needling is emphatically the best available treatment for carcinoma of the lip, anterior part of the tongue, and buccal mucosa. For the back of the mouth and tongue, and the pharynx (sites inaccessible to interstitial therapy), radium beam and super-voltage x-ray therapy are the most promising methods.

(3) Importance of careful planning and technique

It is at this point proper to insist that radiation must be carried out with the same precision of planning and technique as the more spectacular radical surgery. The responsibility is just as great: the success or failure of the procedure means life or death to the patient, as a rule he will have no second chance.

Before considering the treatment of the various sites individually, it will be convenient to give some general account of interstitial radiotherapy. In the following sections will be found a brief reference to the underlying theory, simple rules for planning and calculating dosages, and a description of the technique of radium needling.

2. INTERSTITIAL RADIUM THERAPY: THEORY

(1) Physical basis

The radium atom disintegrates, forming an atom of radon gas. This in turn breaks up, liberating the electro-magnetic radiations—*gamma-rays*—used in therapy. In radium therapy, the radon gas is enclosed with its parent radium in air-tight cells, so that the decaying gas is constantly replenished and the issuing radiation remains uniform in strength. For radon therapy, the gas is withdrawn from the radium, sealed in glass or gold capillaries and used alone. Fifty per cent is lost by breakdown in 38 days, and in

Source of
γ-rays

Radium

Radon

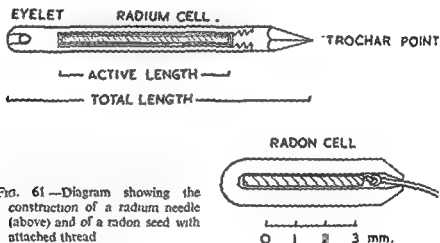


FIG. 61.—Diagram showing the construction of a radium needle (above) and of a radon seed with attached thread

14 days the radiation has dropped practically to zero. The amount of radiation, whether from radium or radon, is measured by its ionizing effect upon air, and is expressed in "roentgen units", usually written as "r". A radium needle (Fig. 61) consists of a platinum tube containing a known weight of radium salt, and threaded at the ends for the attachment of an "eye" and a "point" which when screwed into place seal the tube. The effect of the platinum is to retain other unwanted radiations, transmitting almost pure *gamma* radiation. The value of these rays in treating cancer lies in their selective destructive action on cells in mitosis.

(2) Comparison of radium and radon

The relative merits of radium and radon in treatment may be summarized thus. Radon seeds or needles can be supplied to almost any specified dimensions and linear density (radon content per unit length), and this gives great adaptability. On the other hand, their preparation must be put in hand several days in advance; and once made they do not allow of any change of plan should fuller inspection of the lesion, in the theatre, reveal unsuspected extensions or complexities. Thus, when the extent of the disease is hard to assess

Convenience
of radon

pre-operatively, a generous selection of radium needles offers greater possibilities.

Radon seeds have little intrinsic value, and may therefore be used in out-patients. If necessary, too, they may be left permanently in position (though undesirable results sometimes follow). *Cheapness*

In brief, when an adequate selection of radium needles is available, they have many advantages; but when radium is in short supply or not available, radon is an excellent alternative.

(3) Paterson and Parker system of dose calculation

What has become known as the "Manchester system" can be found fully described in the publications of Paterson and Parker to which reference

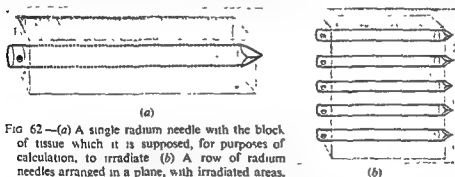


FIG 62—(a) A single radium needle with the block of tissue which it is supposed, for purposes of calculation, to irradiate (b) A row of radium needles arranged in a plane, with irradiated areas.

is made in the bibliography. A sufficient account of the system will now be given to enable most tongue and mouth implants to be carried out satisfactorily. The method consists in breaking down the area or volume to be

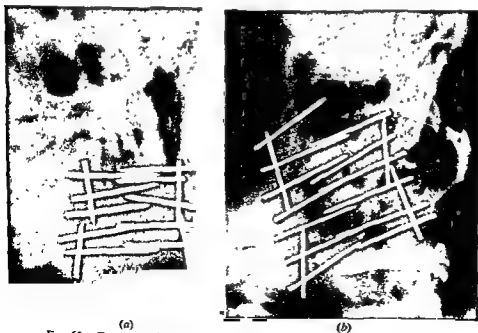


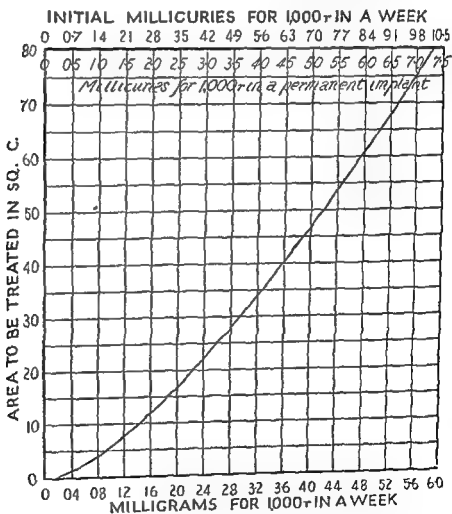
FIG 63—Examples of single-plane implants (a) for the lip, (b) for the cheek.

needed into simple geometrical shapes, for which a fairly uniform dose distribution can be worked out by following simple rules and using the graphs reproduced in simplified form here.

*The single
radium
needle*

*The row of
needles*

It is convenient, and sufficiently accurate, to regard a radium or radon needle as irradiating a cylindrical or rectangular block of tissue, of which it forms the long axis (Fig. 62 (a)). The block is as long as the needle minus its dead ends, and 1 centimetre in diameter. Dosage values are worked out for the surfaces of the block, and therefore represent *minimum* values. A row of parallel needles, spaced 1 centimetre apart, may similarly be considered as irradiating a block of tissue 1 centimetre thick and coextensive with the needles minus the dead ends (Fig. 62 (b)). To compensate for falling-off of intensity at the ends and sides of the block, the needle-ends may be "crossed" with others, and all the marginal needles should contain more radium per



GRAPH I.—AREA—Giving quantities of radium or radon required for plane implants. (See text.)

unit length The whole arrangement now constitutes what is known as
according to the following rules.

*"Single-plane
implant"*

(a) *Single-plane implant*

(i) *Quantity required*.—Determine from Graph 1—Area, the number of milligrams needed to deliver 1,000 r units in a week to the area under consideration. Multiply this by 6, 7 or 8, and so on, according to the total dose it is deemed safe and desirable to give.

(ii) *Distribution*.—Arrange the quantity so obtained over the area as follows (Table II).

TABLE II.—TABLE FOR SINGLE-PLANE IMPLANTS

Area of implant	Proportion of total radium arranged round periphery
0-25 sq cms	Two-thirds
25-100 sq cms	Half
Over 100 sq. cms.	One-third

Note The remainder of the radium is evenly distributed throughout the area.

(b) *Two-plane implant*

For flat blocks of tissue more than 1 centimetre thick, as for example in massive lip or cheek growths, two parallel planes of needles may be used, spaced 1, 1½, 2 or even 2½ centimetres apart. The amount of radium for each plane is given by the same Graph 1, but a "correction factor" is used to increase the dosage, which would otherwise be low midway between the planes. This factor is as follows.

TWO-PLANE CORRECTION FACTORS

Planes 1 cm apart				
1½ cms	.	.	×	1.0
2 cms	.	.	×	1.25
2½ cms	.	.	×	1.4
	.	.	×	1.5

Calculation is more complicated when thicker or less regular blocks of tissue are to be irradiated, and it is here that the co-operation of the physicist

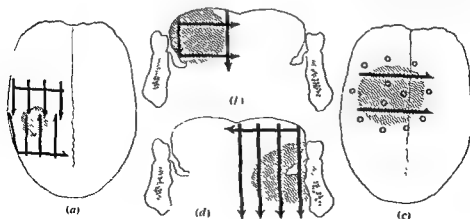
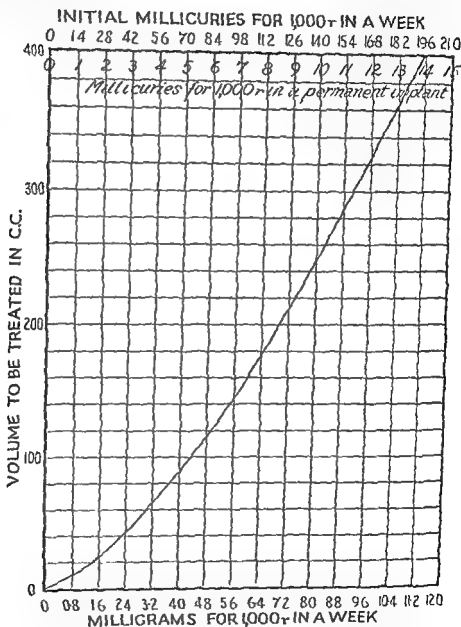


FIG 14.—Method of implanting various types of tongue carcinoma (For details see text)

is very welcome. The block of tissue may be treated as a cylinder, a sphere, a cube or cuboid, a cone, a prism and so on. The method is as follows.

(c) *Volume implant*

(i) *Quantity required.*—Read off Graph II—Volume, the milligrams required for the given volume, and multiply according to the dose it is proposed to give, as before.



GRAPH II.—VOLUME.—Giving quantities of radium or radon required for volume implants (See text.)

(ii) *Distribution.*—The quantity thus obtained is distributed throughout the volume, which is visualized as consisting of “rind” and “core” (see Table II).

Some diagrammatic examples illustrating the application of these “geometrical” implants to tongue and mouth sites are shown in Fig 64.

TABLE III.—TABLE FOR VOLUME IMPLANTS

Shape	Distribution
Sphere . . .	rimd: $\frac{1}{2}$; core: $\frac{1}{2}$
Cylinder . . .	rimd: $\frac{1}{2}$; each end: $\frac{1}{4}$; core: $\frac{1}{2}$
Cuboid . . .	each side and each end: $\frac{1}{4}$; core: $\frac{1}{2}$
Rectangular block . . .	the block may be treated as a series of parallel planes, the outermost pair containing each 3 parts, and the inner planes each 2 parts, of the total radium.

Note: The radium for the core should be spread evenly through the volume in each case, and *not* concentrated at the centre.

3. INTERSTITIAL RADIUM THERAPY: PRACTICE

(1) Preparation and anaesthesia

Extraction of carious or infected teeth should be carried out at or before operation, but time should not be wasted in prolonged dental treatment. Dental extraction after radiation is dangerous as the trauma sometimes precipitates necrosis of the jaw.

Anaesthesia must be of the highest standard, with careful selection of agents according to the type of patient, situation of the lesion, and whether or not diathermy is to be used. Faultless endotracheal intubation is necessary to avoid trauma to the malignant lesion. The constant danger of inhalation of foreign matter requires vigilance and skill. For intra-oral work the throat must be carefully packed, preferably with Vaseline gauze, and a sucker must be available. General anaesthesia has the great advantage of providing the fullest exposure of the lesion.

(2) The implant

Radium needles are prepared by threading them with "strings" of stout *Preparation of needles* linen or twisted silk. Each length of "string" should measure about 20 inches, and its ends should be burnt through in a spirit-lamp flame rather than cut, to prevent fraying (Fig. 65 (a)). Care must be taken not to damage the "string" when grasping the needle with the forceps in order to implant it. The needles must be implanted accurately, and for this purpose a steel measure and a pair of dividers are useful. These are shown in Fig. 66 which illustrates the apparatus needed for the operation. As the needles are inserted, each one must be *Necessary apparatus* securely fixed in place. For mucosal implants this usually means individual suturing by one of the methods shown in Fig. 65. (1) The "string" itself may be threaded on to a spear-pointed needle of Mayo type. This is then made to pick up a substantial bite of firm tissue, and the two "strings" are tied with a reef-knot (Fig. 65 (c)). (2) Alternatively, a separate stitch of finer thread or silk, preferably of contrasting colour, is stitched through the tissue and then tied over the "string" (Fig. 65 (b)). This method makes removal rather easier. After checking the number of "strings" and tying them together, they are stitched to the skin for security or covered with several small strips of elastic strapping. If the implant is a cutaneous one, the needle punctures may be sealed with a liquid adhesive, to which a gauze dressing is allowed to adhere. *Dressing*

Radon seeds, if their subsequent removal is planned, are ordered with threads attached. The seeds may be inserted by boring a hole with a hydrocele *Seed implants*

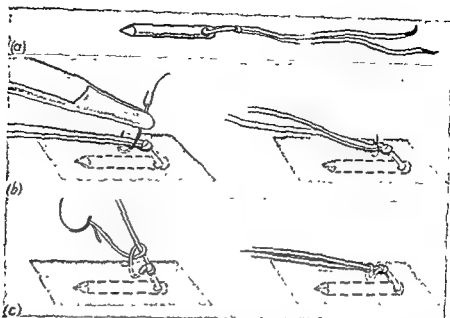


FIG. 65—(a) A radium needle prepared with its string. For mucosal insertion the knot is tied hard on to the end of the needle, whereas for subcutaneous insertion a gap of a few millimetres is left. Note the ends of the string which are charred to prevent fraying. Beneath are shown two alternative methods of securing the needle in position; (b) by means of a separate stitch; and (c) by using the string of the needle

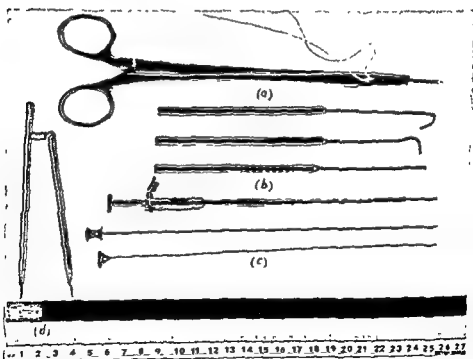
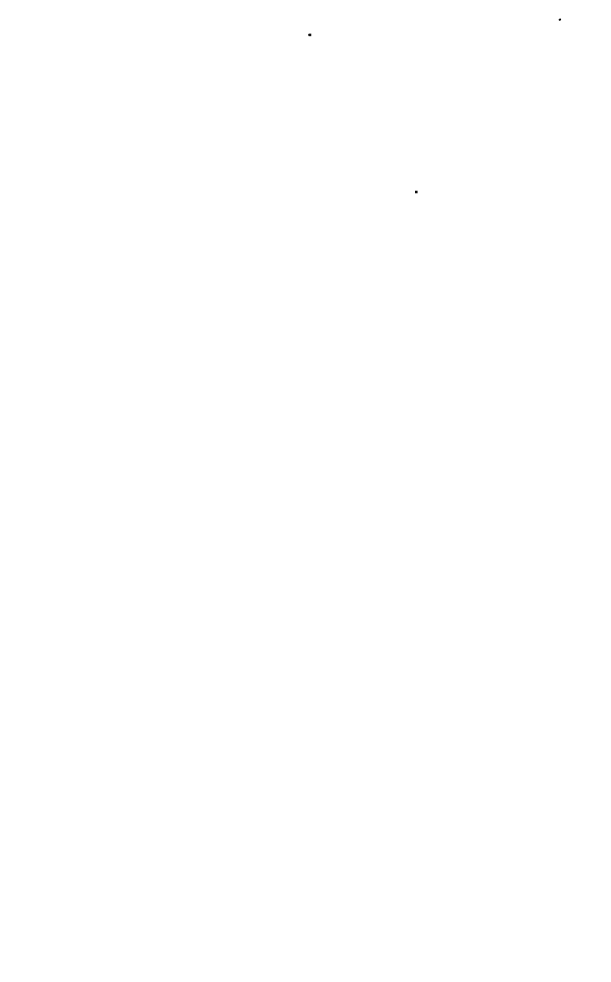


FIG. 66.—Apparatus for radium or radon implants. The photograph illustrates: (a) a threaded needle in holding forceps; (b) 3 "pushers" of different angles for "pushing" the seed into and (c) radon-sealed introducer consisting of a cannula into which the seed is dropped and which the seed can be pushed out of the cannula into the tissue.



trocac and thrusting the seed into the tunnel thus made. The point of a straight needle serves to push the seed into place. Alternatively, one of the many specially designed "guns" illustrated may be used. Fig. 66 illustrates one of the simplest of them. The seed introducers (Fig. 67), which are

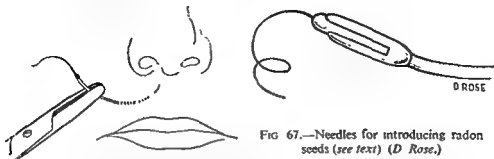


FIG 67.—Needles for introducing radon seeds (see text) (D Rose.)

surgical needles armed with a spring clip to hold the seed, may be used. They are held in a needle-holder, and passed through the tissue. When the seed lies in the desired position, it is dislodged by a tug on its string, and left behind as the needle is pulled out.

(3) Dosage

Dose calculation has already been described, and it has been pointed out that the help of a physicist is very desirable. When, for example, skiagrams are taken of the needles *in situ*, irregularities may be disclosed which make re-calculation essential; and some insertions are so complicated that usefully accurate arrangements can only be devised by those skilled in physics and mathematics. In deciding the limits of safe dosage, however, physics must be subordinated to clinical experience. The final decision as to when the needles shall be removed will be influenced not only by the particular tissues being treated, but by the individual susceptibility of the patient as evidenced by his reaction, by his physical condition, his age and *habitus*, alcoholism, proximity of radio-sensitive structures such as cartilage and bone, the presence and degree of sepsis, and so forth.

Clinical
assessment
Factors
influencing
safe dosage

(4) Management and after-care

The insertion of needles or seeds may be followed by considerable swelling, and if there is much evidence of sepsis full doses of penicillin may be given for its control. In most instances the swelling starts to subside after 3 or 4 days, if not, it may in rare cases be necessary to consider removal of the needles a day or so earlier than was originally planned. At or soon after the removal of the needles, the lesion will be seen to be covered on its mucosal part with a uniform "radium film" of yellow colour and smooth and clean appearance (Plate I (a)). This may also appear on neighbouring parts which have been in contact with the needled area. The film, which is composed of fibrinous exudate and dead epithelium, disappears slowly, often taking some weeks to vanish finally. Skin, when the needles are removed, will have a red and inflamed appearance (Plate I (b)), and there may be discharge of purulent-looking material from the needle holes, with crusting of any ulcerated area. It must be emphasized that the discharge consists largely of sterile necrotic tissue, and does not necessarily imply serious infection. If, however, local

Filmung

Debris

Infection

infection is severe, boric fomentations may be advisable for 1 or 2 days, and thereafter dusting with penicillin and a sulphonamide powder will cause rapid healing. A non-greasy cream of penicillin is very useful in preventing the adhesion of opposed mucosal or skin surfaces. Irritant applications should not be used, as they will only aggravate the reaction and delay healing.

4. THE SITES CONSIDERED INDIVIDUALLY

(1) The lip

(a) Choice of treatment

Surgery

The drawbacks of surgery have been mentioned already, and it has been made clear that the authors consider radiation the treatment of choice. This method will therefore be discussed first. For the benefit of those to whom neither radium nor radon is available, the surgical methods will then be described.

Radium moulds

Radiation of the lip may be by radium plaque (mould) or by interstitial therapy. The choice is largely a matter of personal preference. Some large growths may be more suitable for mould than for implant, but on the other hand the quantities of radium required are greater and the physical calculations more complicated. The present description will therefore be limited to interstitial methods, and those interested in the alternatives are referred to the works of Paterson and Parker in which a full account will be found.

(b) Radium needling

*Removal of teeth**Type of implant*

Local or general anaesthesia may be used, but before inserting the needles it is wise to extract any teeth which impinge on the lip, especially if they are carious or much infected. Radium or radon may be used, and the following remarks apply equally to both. A one-plane implant is used for thin growths; and for those more than 1 centimetre or so thick the two-plane implant is suitable. A wide area of the lip is needled, and a usual arrangement of the radium is shown in Fig. 60 (b). Note the use of short needles to allow for the convexity of the jaw, and the overlapping of the "dead ends" of the needles. Each "string" is sewn in place by a silk stitch, and the whole is covered with small pieces of elastic strapping after the threads have been tied together for security. If the growing beard tends to push the strapping away, additional layers should be laid over it as necessary, and every effort made to avoid dislodgement. Provided the insertion is accurate, and the dose distribution uniform, it is safe to aim at a total of 7,000 to 8,000 roentgens in 7-8 days.

*Dressing**Dose*

(c) Surgical excision

The area to be excised must be wide, and any temptation to restrict the clearance should be resisted. Fig. 68 shows one of the most generally useful methods of closing the gap thus caused, the stages of the operation being carried out as follows.

(i) *Anaesthesia*.—General anaesthesia is induced and the trachea intubated. The throat is securely packed to prevent inhalation.

(ii) *Excision of the growth*.—This includes the whole thickness of the lip, and leaves a roughly square or oblong defect. Bleeding is controlled by compressing the lip between finger and thumb until the severed vessels have been grasped with haemostats.

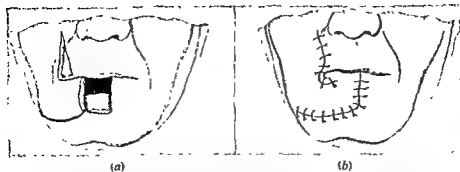


FIG. 68.—A method of repairing the defect resulting from excision of a growth of the lower lip. The gap illustrated is about 3 centimetres wide, and in actual practice the mobilization depicted is carried out bilaterally.

(iii) *Preparation of the flaps.*—The gap may be closed by sliding forward flaps on one or both sides, and a red margin may be provided by turning down part of that of the upper lip. The upper lip is correspondingly narrowed by taking out (bilaterally if necessary) wedges as shown in the diagram (Fig. 68).

(iv) *Closure.*—The mucosa is sutured first, using a fine catgut with the knots tied outside the mouth. Every effort should be made to complete this closure, as primary healing is then much more certain. A few stitches may be inserted in the muscle layer, and the skin is then sutured with fine interrupted sutures of nylon or silk. Finally, the red margin is fitted to the free edge of the flaps. *Accurate mucosa closure necessary*

Where the ablation includes the whole lower lip, the reconstruction devised by Syme may be used. Lateral flaps are fashioned by two long symmetrical incisions starting in the midline and curving downwards and backwards across the submandibular triangles, then outwards to end near the mandibular angles. The flaps thus outlined are freed, and rotated upwards and inwards to form the new lip. They are sutured together in the midline, and anchored by reattachment to the fixed median wedge of skin which remains at the point of the chin.

If the defect is too large to be closed in this way, and especially if the excision encroaches on the cheek, it is better to suture the skin margins of the defect to the mucosa, and deal with the case by means of pedicle flaps by stages.



FIG. 69.—Skigram showing involvement of the mandible by a carcinoma of the floor of the mouth. If not too extensive, such a lesion should be treated by resection with the affected bone.

(2) The tongue, mouth floor and alveolus

(a) Choice of treatment

*Indications
for surgery*

For these sites, as for the lip, interstitial radiation is considered the first choice; but there are certain specific indications for surgery as follows.

- (i) Involvement of the mandible by growth (Fig. 69).
- (ii) Involvement of the whole tongue.
- (iii) Failure of, and recurrence after, interstitial therapy.
- (iv) A painful, indurated sclerotic tongue due to radium overdose may occasionally call for surgical removal, even in the absence of active disease.

(b) Radium needling

*Small
lesions**Large
tumours**Mouth floor*

The illustrations (Fig. 64, page 105) are designed to show some of the commoner types of lesion, and how they may be dealt with. Fig. 64 (a) represents a small, flat lesion, conveniently treated by a single-plane implant placed just deep to it, and extending beneath 3-4 times the apparent diameter of the growth. Fig 64 (b) shows a growth near the lateral margin of the tongue, for which the whole volume of the organ to the midline is to be treated. A two-plane implant is shown, long needles being placed horizontally in the tongue and the ends crossed horizontally or vertically according to convenience. In Fig. 64 (c) the lesion involves the more solid part of the tongue, and the needles may be placed perpendicular to the surface, treating an imaginary cylinder on end. As in Fig. 64 (d), for a lesion affecting the under-surface of the tongue, and extending on to the mouth floor, it may be necessary to consider the two parts as forming one whole. The volume thus formed may be needled with two horizontal planes, one in the tongue and the other in the mouth floor; or in more advanced cases needles may be inserted vertically, pinning the tongue down to the mouth floor, the cylinder or cuboid thus formed being closed below by needles inserted from the submental region.

(i) *Dosage.*—The tongue is tolerant of radiation, and may be given high dosages. A dosage of 8,000 roentgens in a week is well tolerated, provided the implant is not in contact with the alveolus and the mouth is not in a very septic state.

*Feeding**Cleansing*

(ii) *After-care.*—During the week of treatment, the patient is discouraged from talking and is provided with pencil and paper to make known his wants. Fluid nourishment is given in abundance, if necessary with a rubber-spouted feeding-cup or in some cases a nasal-feeding catheter; and the mouth is kept clean with frequent mouth-washes of Glycothymoline or bicarbonate of soda. The mouth may be syringed very gently with a Higginson's syringe equipped with an aural nozzle, provided that some responsible person is available to undertake this treatment. Penicillin lozenges may be allowed to dissolve in the mouth. Penicillin by injection is advisable when reaction is severe. At the end of the week the tongue will be swollen, and there may be some filming. Saliva will be scanty and ropy, and the submental tissues may be swollen. The cleansing of the mouth becomes easier after the needles are removed, and the syringing should be carried out several times daily, especially after feeds. During treatment, the patient may suffer discomfort from the pressure of the wet threads between the lips. This may be minimized by applying a bland ointment freely to the lips, and by wrapping a piece of Tulle Gras or soft rubber tissue round the threads. If the lesion is at the back of the mouth it is

sometimes easier to draw the threads out through the nostril by attaching them to a catheter passed through the nose. They should not be pulled too tightly round the soft palate. The bunch of threads, tied together for security, may be fastened to the cheek with a stitch or with adhesive strapping.

(c) Surgical excision

(i) Diathermy glossectomy.—The old "set"

operations devised by Syme, Whitehead, Kocher and others, and practised so brilliantly by Butlin, have given way to diathermy excisions modified to suit the individual case. The steps of the method are as follows.

Anaesthesia: general anaesthesia is induced, the trachea intubated, and the throat packed securely.

Ligation of the external carotid artery: this may be performed on one or both sides as a preliminary if the operation is to be extensive.

Excision: the mouth is propped well open and the healthy part of the tongue seized with forceps. The diathermy needle is made to divide the tongue slowly, using a small cutting current. Bleeding is slight, and is readily dealt with by picking up any vessels and applying the coagulating current to the haemostats. *No attempt is made to cover the raw surface or close the gap in the tongue.* The after-treatment is the same as already described in the section dealing with interstitial therapy. The post-operative course is

*Slow cutting
lessens
bleeding*



FIG 70.—End-result of a diathermy hemi-glossectomy performed for recurrent carcinoma of the tongue. Note the clean healing, the satisfactory amount of protrusion and the free view into the pharynx, where the tip of the epiglottis is seen.

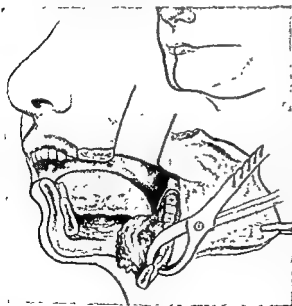


FIG 71.—Excision of the mandible. The inset shows the incisions which may be used. The larger drawing shows the lesion isolated and the jaw prepared for the second (posterior) section.

remarkably painless, and when the raw surface has epithelialized movement and function recover to a surprising degree. Fig. 70 shows a case in which the left side of the tongue has been removed in this way, leaving a clear view into the pharynx. The amount of protrusion is shown, and speech and eating are reasonably good.

(ii) *Removal of the mandible.*—Anaesthesia, intubation, packing and preliminary ligation of the external carotid artery are carried out as above. Two of the most useful incisions are shown in Fig. 71 (inset). The longer incision is intended for cases with involvement of the mouth floor extending on to the alveolus, and, starting in the midline of the lower lip, it extends down to the submental and submandibular region of the affected side. If desired, it can be extended still further and used for the carotid ligation. The resulting flap is freed from the jaw and the submandibular tissues, and retracted outwards. The mandible is next divided with a Gigli saw distal to the lesion, leaving 2–3 centimetres of clearance. If the division is made near the symphysis, it is an advantage to leave the genial tubercles, because the conservation of their muscle attachments gives better function to the tongue. The diseased portion of the mandible is then grasped with lion forceps, and retracted laterally. The tongue (if necessary) and the floor of the mouth are gradually divided with the diathermy, and the mass is further rotated outwards to give access to the posterior part of the mandible. The site for the second section of the bone is selected and the bone stripped of periosteum. If the submandibular region has been well cleared, by freeing the fatty tissues with the lymph glands, and the salivary gland from the underlying muscles, the specimen is now attached only by the bone. This is divided, usually with a Gigli saw, and the specimen removed.

Closure: the first step is to cover the bone-ends. This usually presents no difficulty, the mucosa being sufficiently mobile to be drawn over the cut surface. Next, the mucosa of the mouth is carefully and completely closed, using interrupted stitches of fine catgut with the knots tied outside the mouth. Good functional result depends largely on accurate closure. In some cases intermediate stitches may be inserted, especially in the lip, to secure the muscle layer. The skin is now closed with interrupted stitches of fine nylon or silk.

If desired, the two ends of jaw may be kept apart by attaching at this stage some type of dental splint. This may be prepared beforehand and wired on to any teeth present (Fig. 72 (a)) or if the patient is edentulous, external fixation may be obtained with bone pins using the Clouston-Walker or similar splint (Figs. 72 (b) and 73). This enables bone grafting to be undertaken at a later date, if the case appears to warrant it. In many elderly patients or in advanced cases, however, further operative procedures are undesirable, and sufficiently good function and appearance are usual even without grafting.

The additional incision, splitting the cheek horizontally outwards from the angle of the mouth, is designed to give access to growths far back in the region of the faucial pillar, involving the mandible in the region of its angle.

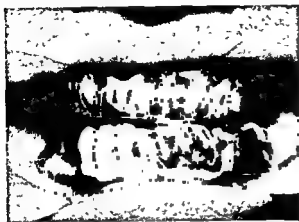
(3) Check mucosa

The cheek may readily be needled, usually from within, with a single-plane implant; or, in the posterior thicker part, with a two-plane implant. In many

*Accurate
mucosal
closure*

*Splinting
jaw to
prevent
displacement*

*Repair of
bony defects*



(a)



(b)

FIG. 72.—Excision of the mandible. Methods of fixing the residual portion to prevent displacement. (a) A dental cap splint prepared and cemented in before operation; the two portions will be wired together. (b) Anchorage by means of a bone pin fixed to a plaster head-band.



FIG 73—A Clouston-Walker splint applied to a model (*Apparatus shown in Figs. 72 and 73 from Down Bros. and Mayer and Phelps.*)

*Problem of
alveolar
involvement*

cases the upper or lower alveolar mucosa is involved, and the following alternatives are then available. (a) Interstitial needling, the needles being inserted under the muco-periosteum if they are sufficiently slender and tearing does not follow. (b) Diathermy coagulation of the alveolar portion of the growth, with a view to excision later, if the response of the cheek to radiation is good. (c) Radical surgical excision on the lines indicated above, if the alveolar involvement is severe. Unfortunately, excision of the cheek tissues usually leaves a defect not capable of simple closure. The skin edges should be stitched to the mucosa, and the gap later closed in stages by a pedicle flap.

(4) The hard palate

(a) Radiotherapy

Small, flat growths of the hard palate may be very suitable for mould treatment. The mould is constructed after the manner of an upper denture, of composition or acrylic resin. Fig. 74 shows such a mould with a tray to take needles or seeds and provided with a screw-on lid. It is designed to give a moderately uniform dose at a distance of 0.5 centimetre. As a refinement, the tongue may be afforded some protection by the incorporation of a lead shield between it and the radium source, or by "biting blocks" which serve to keep the tongue as far away from the radium as possible.

*Radium
moulds*

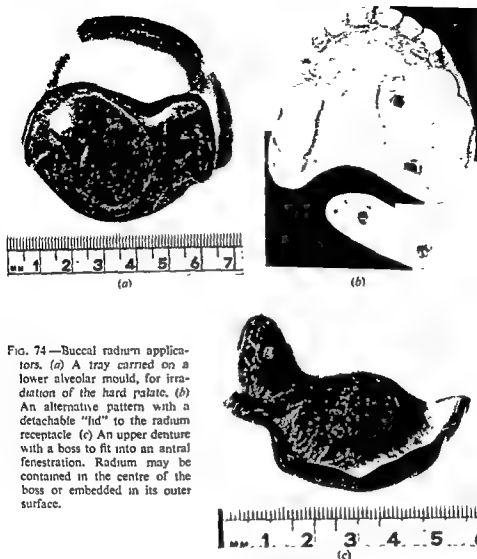


FIG. 74—Buccal radium applicators. (a) A tray carried on a lower alveolar mould, for irradiation of the hard palate. (b) An alternative pattern with a detachable "lid" to the radium receptacle (c) An upper denture with a boss to fit into an antral fenestration. Radium may be contained in the centre of the boss or embedded in its outer surface.

(b) Surgical excision

In more extensive cases, particularly when the bone is grossly involved, diathermy coagulation and excision are indicated. This is performed as described in the section dealing with fenestration of the maxillary antrum (p. 118) and results in a defect of the bony palate requiring a dental obturator.

(5) Soft palate, fauces, tonsil, back of tongue and pharynx

Except in the case of very minute lesions, growths in these situations are unsuitable for surgery or for interstitial radiation. Very early lesions may sometimes, however, be excised by diathermy, or implanted with radium or radon. The two operations of transhyoid pharyngotomy and lateral pharyngotomy, originally introduced to enable early growths to be resected, are still sometimes employed to provide access for radium or radon implantation. The best results are, however, obtained with telerradium, or with a combination of this and x-rays. When telerradium is not available, high-voltage x-rays alone may be used. In brief, the method is to irradiate the lesion through

Pharyngotomy

Radiation

a number of ports, including any portion of



FIG 75—The Westminster Hospital 4-Gramme Telerradium unit applied to a case of carcinoma of the tonsil. The standard applicator has been so constructed that when treatment is given through the three fields in rotation, the dosage and distribution of radiation in the neighbourhood of the lesion can be accurately determined from prepared isodose diagrams.

chosen for the nearer ports because of the better quality of its radiation, and x-rays for the more distant because of the greater depth dose. An example of such a plan of treatment is illustrated in Fig. 75.

(6) Maxillary antrum

(a) Choice of treatment

Malignant disease of the antrum is amenable to radical surgery, and to more conservative surgery combined with radiotherapy. The combined method is far more flexible, and can be adapted to almost all cases, including the large number in which "radical" excision of the maxilla is impossible. In the authors' opinion, the classical operation of Fergusson is a mutilating procedure giving results no better than the more conservative method. The latter will therefore be

described first, and an account of Fergusson's operation will follow.

(b) Fenestration of the maxillary antrum

(i) *Principles and purpose.*—This operation consists in principle of making a hole in the hard palate for the three-fold purpose of (a) providing drainage to the usually septic mass within the antrum, (b) permitting diathermy removal of the main mass of growth, and (c) affording a window through which radium can be inserted and subsequently the results of the treatment watched, and any signs of recurrence readily detected. Operation is in most cases preceded by a preliminary course of x-ray therapy.

(ii) *Technique* (Fig. 76 (a) and (b)).—In the pre-operative period the patient's teeth should have received attention and any blood deficiency should have been made good. A general anaesthetic is given, and an endotracheal tube passed either through the nostril of the sound side or through the mouth. The throat is packed securely to prevent inhalation. Non-inflammable anaesthetic gases must be used. The patient lies on his back, with a sand-bag under the shoulders so that the head is slightly hyperextended. The surgeon

Anaesthesia

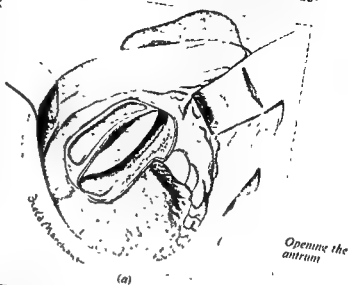
Position of patient

MAXILLARY ANTRUM

119

may stand facing the patient, or may seat himself at the end of the table and work over the head of the patient. A head-mirror and good light and a suction apparatus are essential.

Cheek and tongue retractors of bakelite or Perspex are valuable, but good substitutes may be made by covering long haemostats with tightly fitting rubber tubing. The area of palate to be removed, which should include any part involved by growth but should spare, if possible, the soft palate, is outlined by cutting very slowly with the diathermy, using a small cutting current. The bone of the palate and, if necessary (as is often the case), of the alveolus, is cut through with a few taps of a narrow osteotome, and the diathermy needle is then passed into the antrum. It is swept round the cavity very slowly, using a weak coagulating current; this movement is continued until the more or less cylindrical mass of tissue, including the disc of palate, can be lifted away. The posterior palatine vessels may require to be picked up and coagulated. The antrum, and possibly the nose, will now be displayed to view, and an attempt should be made to clear away the gross mass of the tumour tissue, using the diathermy loop and a very slow, patient movement, with small current. If the outer nasal wall is diseased, this is also removed together with the turbinates. Should the ethmoid cells be infected, they are opened up and drainage is provided for. When a sufficient clearance has been made, the cavity may be packed tightly with gauze wrung out of saline solution at 120° F., or with gauze soaked in cold 20-volume peroxide of hydrogen. In this way, bleeding can usually be



Opening the antrum

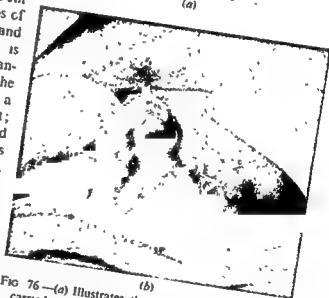


FIG 76—(a) Illustrates the fenestration operation as carried out for a carcinoma of the right antrum. The nasal cavity also has been opened. (b) Showing healing after fenestration. The opening in this instance rather small, is covered by the patient's denture and is antrum

symptomless

Haemostasis

arrested, but large vessels may have to be grasped with haemostats, which are then touched with the coagulating current. Plugging with gauze soaked in topical thrombin may be valuable if the bleeding persists. Finally, the cavity is insufflated with penicillin and sulphathiazole powder and packed firmly with Vaseline ribbon gauze. The throat is carefully sponged dry as the anaesthetist's pack is removed, and the patient returned to the ward lying on his side, with the mouth towards the pillow. He may be placed sitting up as soon as he recovers consciousness, and is allowed out of bed the following day. The pack is removed by irrigation after 24 or 48 hours, and thereafter the cavity is left open for drainage except at meal-times, when it is filled with a tampon made by surrounding a ball of cotton-wool with gauze, or by a dental plate made of plastic. Provided that the soft palate has been left intact, the operative defect can later be closed with a dental plate, and speech and eating then become almost perfect.

After-care

(iii) *Pre-operative and post-operative radiotherapy.*—This type of limited operation must be combined with suitable radiotherapy. Pre-operatively, x-ray therapy is often given, especially in advanced cases. After operation, several alternatives are possible, namely x-ray therapy; a cavitary mould carried on an upper "denture" and supplied with radium or radon over its surface (Fig. 74 (c)), or a central radium or radon source fastened in the middle of the cavity with the object of delivering a better depth-dose to the walls. Occasionally, the residual growth has been needed interstitially through the cheek, usually when the latter is much involved.

(iv) *Prognosis.*—Cures are claimed for some 50 per cent of cases; but it must be emphasized that even if cure is not obtained the palliative value is enormous. It is hardly ever justifiable to refuse treatment for an antral tumour. Survivals of 20 years, free from disease, have been recorded.



(c) *Excision of the maxilla (Fergusson's operation)*

The bony attachments of the maxilla are illustrated in Fig. 77, and the method of excising it is as follows. Pre-operatively, the teeth receive attention, and the blood is restored to normal. The patient is anaesthetized, and the trachea intubated through either the sound nostril or the mouth. The throat is securely packed, and the patient placed on his back with the head well extended by a sand-bag under the shoulders. As

*Anaesthesia**Position of patient*

FIG. 77 —Excision of the maxilla. Skull showing the bone cuts needed to free the upper jaw: (a) through the nasal process; (b) through the malar attachment; and (c) through the alveolus and palate.

in the fenestration operation, a sucker is essential, and diathermy is a valuable ally.

The surgeon faces the patient and, first turning the head to the opposite side, ligates the external carotid artery. The head is then straightened, and the incision made as in the illustration. The soft tissues are turned back as a flap, diathermy being useful as it diminishes bleeding. The bony attachments of the maxilla are cut through in the following order: (i) the nasal process, using sharp narrow-bladed bone forceps; (ii) the malar articulation, using a thin osteotome, and guarding the orbital contents with a retractor. The cut passes into the inferior orbital fissure. Alternatively, the malar bone itself may be divided with a Gigli saw if this seems necessary to clear the growth. In some cases it may be possible to conserve the orbital floor, which markedly lessens the resulting deformity. *Carotid ligation*
Flap
Detachment of bone

The mouth is now propped widely open, and the upper incisor teeth, if present, are extracted. With the diathermy the hard palate is divided in the midline down to the bone, and separated from the soft palate right out to the tuberosity. A Gigli saw may now be passed through the nose and out at the mouth, or if a thin enough bone forceps is available it may be used. The palate being thus divided, an osteotome is inserted into the gap and gently levered to loosen the maxilla. If necessary, the attachment to the pterygoids may be divided by retracting the skin flap and inserting the osteotome. If the jaw is now grasped with lion forceps and persistently rocked it will separate. If it is very much diseased it may have to be removed in sections. Haemostasis is secured by pressure packing with gauze wrung out of hot saline solution (120° F.) or cold hydrogen peroxide. Vessels are seized and coagulated where necessary. The skin flap is sutured into place, and the cavity packed through the mouth with a Vaseline gauze roll. The subsequent treatment of the palatal defect, and the management of the patient, are as described in the section dealing with fenestration of the antrum. *Haemostasis*

(7) Cervical lymph glands

Surgery is the treatment of choice for lymph-gland metastases of squamous-cell carcinoma which, unlike the primary lesions, usually respond poorly to radiotherapy. The most satisfactory procedure is the block dissection, a detailed account of which follows.

(a) Block dissection of the neck

(i) *Indications and contra-indications.*—By “block dissection” of the neck is meant the removal of the sheath of deep cervical fascia from the horizontal ramus of the mandible to the clavicle and from the midline anteriorly to the anterior border of the trapezius posteriorly. Such excision carries with it the various groups of lymph glands in the submaxillary and anterior triangles of the neck. For a complete excision of the lymph glands it is necessary to remove the sternomastoid muscle, the internal jugular vein, and the omohyoid and digastric muscles. *Definition*

The operation is indicated when the following conditions prevail. (1) The primary lesion in the mouth or lip must be healed and there must be a reasonable prospect of permanent control of the disease. (2) The cervical lymph glands must be enlarged and clinically malignant. (3) The glands *Indications*

must be not only removable, but strictly operable. (4) The general health of the patient must permit a major operation. (5) There should be a normal expectation of a 5-year survival. All experienced surgeons who practise the block dissection are agreed that if the condition of the lymph glands is not strictly operable, the results of the operation are disappointing and local recurrence is inevitable.

*Contra-
indications
Prophylactic
block not
justified*

The contra-indications to the operation must be emphasized. They are as follows. (1) Absence of enlarged glands. The so-called "prophylactic" block dissection has not proved a justifiable procedure, though it may have a place in cases in which follow-up difficulties make regular examination of patients impossible. (2) Old age and poor general health, which increase the operative risks very considerably. Age by itself is not an absolute contra-indication, as some patients are "surgically" young in the seventh decade. (3) Fixation of the lymph glands, however slight, to the vascular sheath or the pre-tracheal fascia, precludes a complete removal of the malignant tissue and rapid recurrence is the rule.

*Two stages
necessary*

(ii) *Bilateral block dissection.*—This is indicated when clinically malignant and operable glands are present on both sides of the neck. An interval of at least 3 weeks, but preferably 6 weeks, should elapse between the two operations; otherwise the venous return from the brain is impaired after the removal of the remaining internal jugular vein, and cerebro-vascular disturbances follow the operation. The need for a bilateral block dissection is indicated in patients with a lesion of the central part of the tongue or floor of the mouth which has responded well to treatment, and when the glands are quite mobile; it is nevertheless of grave prognosis and is but seldom justifiable. In the case of lesions of the nasopharynx, soft palate and uvula, the enlargement of the cervical lymph glands is more often bilateral than unilateral. In such cases, surgical treatment to the neck is not advisable, as the glands spread widely and are hardly ever removable; in such cases radiotherapy is the treatment of choice.

*Local
anaesthetic
undesirable*

(iii) *Operative technique—Anaesthesia:* although the operation has been performed many times under local or regional anaesthesia, the method of choice is general anaesthesia. It is not desirable to infiltrate the cellular tissues of the neck with anaesthetic solution when there is malignant disease in the vicinity of the vascular sheath. Infiltration deep to the platysma is practised by some surgeons with the object of obtaining a bloodless field during the early stages of the operation. Premedication with morphine and scopolamine, and induction with intravenous Pentothal Sodium, followed by intratracheal gas and oxygen, is the usual method.

Position of the patient. a small sand-bag is placed under the shoulder; the head is rotated to the opposite side and slightly extended, but not more than slightly, as otherwise the cervical veins become distended with blood.

Incision: a curved incision is made; it begins behind the mastoid process at the posterior edge of the uppermost part of the sternomastoid muscle and border of the midline anteriorly. The second incision starts at the junction of the descending and horizontal portion of the first incision and extends downwards and backwards

to the clavicle at the posterior edge of the sternomastoid muscle (Fig. 78). This incision, described and practised by Sir Thomas Dunhill, has the advantage of outlining three flaps which by reflexion expose fully the posterior, sub-

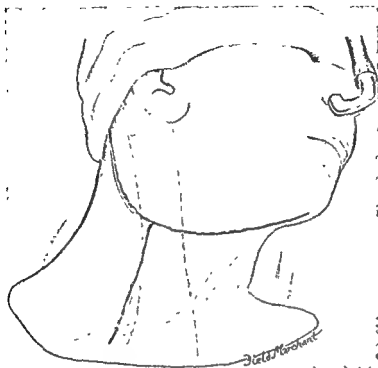


FIG. 78.—Block dissection of the neck. The incisions. The sternomastoid and omohyoid muscles and the mastoid process are indicated.

maxillary and anterior triangles of the neck; it leaves a supple and almost invisible scar.

Raising the flaps (Fig. 79): the three flaps consist of the skin, subcutaneous tissues and platysma; the dissection goes down to the deep cervical fascia, but does not involve it at this stage. The submandibular flap is raised up to the horizontal margin of the mandible. The posterior flap is raised till the anterior edge of the trapezius is defined. The lower anterior flap reveals the infrahyoid group of muscles to the midline. The external jugular and other superficial veins are ligated and haemostasis is completed.

Dissection: it is the authors' practice to start the dissection below. The deep cervical fascia is incised about one finger's breadth above the clavicle along its medial half; the incision is then prolonged upwards. Both heads of the sternomastoid are divided at the level of the central tendon of the omohyoid muscle just below it. The incision is then prolonged posteriorly to the anterior edge of the trapezius; the divided sternomastoid and the cellular tissue behind it and in front of it are dissected upwards. A few veins—the transverse cervical—require ligation. The internal jugular vein is thus brought into view. It is carefully dissected from the common carotid artery and the vagus nerve is visualized. The internal jugular vein is doubly clamped, divided and ligated. The jugular vein is freed gradually from the carotid sheath and the dissection

*Division of
sternomastoid
muscle*

*Isolation
and division
of internal
jugular
vein*

Division of
omohyoid
muscle

carried upwards, ... tendon and the de
fascia cut with scissors near the ... muscles are expos
up to the upper border of the thyroid cartilage and the superior thyroid v
is ligated. Laterally, the dissection is carried down to the scalene musc
exposing the descending (supraclavicular) sensory branches of the cerv

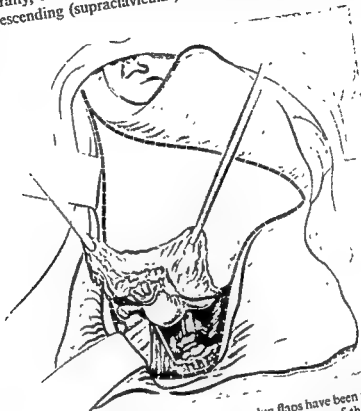


FIG 79.—Block dissection of the neck. The skin flaps have been reflected and the dissection commenced by dividing the origin of the sternomastoid muscle and dividing the internal jugular vein. The vagus nerve and common carotid artery are seen deep to the vein, and the phrenic nerve is lying on the scalenus anticus muscle just lateral to it. The posterior belly of the omohyoid muscle has been divided. The dotted lines show the proposed extent of fascial removal.

Submandibular
dissection

Facial
vessels

Hypoglossal
nerve

Wharton's
duct

plexus, which are divided. At this stage, when the cellular tissue, deep fascia, internal jugular vein and sternomastoid muscle are free up to the upper border of the thyroid cartilage, the submandibular and submental triangles are dissected. The medial or anterior end of the incision in the deep fascia is prolonged upwards and forwards to the level of the mandible and the submental lymph glands, right and left, are excised; the facial vessels are ligated and the anterior belly of the digastric is exposed. The submandibular salivary gland is dislocated posteriorly, so exposing the mylohyoid and hyoglossus muscles. At this stage care should be taken to identify and preserve the hypoglossal nerve (Fig. 80). Most of the submandibular salivary gland is freed and Wharton's duct ligated and divided. Retraction of the mass of tissue thus freed reveals the bifurcation of the common carotid artery and again the hypoglossal nerve as it hooks round the external carotid at the origin of the occipital branch. The mass of glands is separated from the carotid

sheath, the central tendon of the digastric is divided and the posterior belly retracted outwards.

This enables the clearance of the deep fascia and glands to extend up to the transverse process of the atlas, and the styloid process. The line of incision posteriorly is now prolonged upwards along the plane between the posterior

*Division of
posterior
belly of
digastric
muscle*



FIG. 80.—Block dissection of the neck. The dissection completed and the specimen lifted up for the final section of the sternomastoid muscle and jugular vein. The facial vessels are seen, the cut digastric and stylohyoid muscles, and the remaining deep part of the submaxillary gland. The tenth, eleventh and twelfth cranial nerves are depicted, as well as the phrenic. Note that the submental dissection extends across the midline.

border of the sternomastoid and the anterior border of the trapezius, and it is here that the spinal accessory nerve is seen. It is necessary to divide it as, otherwise, the clearance of the upper part of the neck is not complete. The upper end of the sternomastoid is divided and the lower pole of the parotid gland cut across. This exposes the upper end of the jugular vein, which can now be ligated at the level of the atlas (Fig. 80). The division of the vein completes the operation, and the wound is closed completely. A drain is inserted in the posterior flap above the clavicle. A compression dressing is applied so obliterating the space on the side of the dissection.

(iv) *Post-operative course*—Following the operation there is a little difficulty in swallowing, which lasts for 48 hours, some oedema over the mandible for a week and a slight drooping of the angle of the mouth, which recovers in

*Accessory
nerve*

Resulting disabilities

3 weeks. The disability following section of the spinal accessory nerve varies greatly; in some patients, there is hardly any disturbance of function and the raising of the shoulder is normal; in most patients there is weakness of the shoulder, but the range of movement of the arm is near normal. The patient is allowed up on the second day, and hospitalization need not exceed 10-14 days.

(v) *Modified ("submandibular") block dissection.*—In certain cases, particularly with midline lesions of the lips and the floor of the mouth, early but bilateral submandibular lymph-gland involvement is present. When the condition of the patient does not justify a full bilateral block dissection, the following modified procedure may be indicated.

An incision is made extending from the angle of the jaw on one side, curving downwards across the submaxillary region, and up again to the point of the chin. Thence it continues, again curving downwards, to the opposite angle of the jaw. The flaps are dissected upwards, and the submental and submaxillary regions cleared of fatty and lymphoid tissue as above described. The facial vessels are tied on both sides at the lower border of the mandible; the common facial veins are divided near the internal jugular veins and the submandibular salivary glands removed. The wound is closed with a small rubber drain on either side.

(b) *Treatment of inoperable cervical lymph glands*

It has already been stated that the response to radiation of glandular metastases of squamous-cell carcinoma in the neck is not good enough to warrant its use except in cases unsuitable for operation. When the lymph glands, though fixed, are of limited extent, radiotherapy may be undertaken for palliative reasons. Pain may be alleviated, fungation delayed or prevented, and the disease arrested locally for a year or more. Too much should not be attempted; it is difficult, if not impossible, to irradiate up to full dosage the whole lymphatic area of even one side of the neck; and too severe a treatment is prone to lead to painful fibrosis in the fascial layers, thus merely adding to the patient's sufferings.

in implants.—These are suitable for relatively small areas, and may in one or more planes according to the mass to be treated. Long centimetres is a convenient length) promote accuracy in implantation. Dosage should not greatly exceed 6,000 roentgens.

ice applicators—Plaques constructed of Sorbo rubber are fairly like and fit. They may be applied to the neck by means of webbing and buckles, and worn for a convenient number of hours daily. The arranged 2, 3 or 4 centimetres from the skin, and distributed as if it single-plane implant. The uniformity of the dosage obtained is but a large amount of radium is needed and the treatment is pro-

therapy and x-ray therapy.—These methods again are chiefly of use in the treatment of localized swellings. The immediate response of the swelling masses secondary to anaplastic carcinoma of the back of the head and the pharynx is sometimes very gratifying.

BIBLIOGRAPHY AND REFERENCES

- Berven, E. G. E. (1931). "Malignant Tumours of the Tonsil. A Clinical Study with Special Reference to Radiological Treatment". *Acta radiol., Stockh.*, Suppl No XI.
- Butlin, H. (1931). *Diseases of the Tongue*, 3rd ed., by W. Spencer and S. Cade London; Lewis.
- Cade, S. (1948) *Malignant Disease and its Treatment by Radium*, 2nd ed., Vol. 2 Bristol; Wright.
- Godtfredsen, E. (1944) "Ophthalmologic and Neurologic Symptoms at Malignant Nasopharyngeal Tumours". *Acta oto-laryng., Stockh.*, Suppl 59.
- Jacobsson, F. (1948) "Carcinoma of the Tongue. A Clinical Study of 277 Cases Treated at Radiumhemmet, 1931-1942". *Acta radiol., Stockh.*, Suppl. 68.
- Parker, H. M. (1947) In *Radium Dosage; the Manchester System* Ed by W. J Meredith. Edinburgh; Livingstone.
- Paterson, R. (1948). *The Treatment of Malignant Disease by Radium and X-rays*, London; Arnold.
- Pilcher, R. (1937). *Brit med J*, 1, 13.
- (1948) *Proc. R Soc. Med.*, 41, 445.
- Regaud, C. (1923) "Principes du traitement des épithéliomes épidermoïdes par les radiations, application aux épidermoïdes de la peau et de la bouche". *J. Radiol Electrol*, 7, 311.
- Roux-Berger, J. L., and Tailhefer, A. (1933). "Le curage des ganglions du cou dans les cancers buccaux-pharyngés La section systématique du ventre postérieur du digastrique et du stylo-hyoïdien" *Pr. méd.*, 41, 482
- Trotter, W. (1913) Hunterian Lecture on "The Operative Treatment of Malignant Disease of the Mouth and Pharynx" *Lancet*, 1, 1075 and 1147.
- (1920) "A Method of Lateral Pharyngotomy for the Exposure of Large Growths in the Epilaryngeal Region" *J. Laryng*, 35, 289
- (1926) Purvis Oration on "The Surgery of Malignant Disease of the Pharynx" *Brit med. J*, 1, 269.
- (1929) "Operations for Malignant Disease of the Pharynx". *Brit. J Surg.*, 16, 485.
- (Summaries of Trotter's operative results were given by Pilcher, 1937 and 1948)
- Wilson, C. W. (1945). *Radium Therapy, its physical aspects*. London; Chapman and Hall.
- [References to other titles are given under Mouth and Pharynx, Malignant Diseases of, in the Index Volume]

MUSCLE AND TENDON— DISEASES AND INJURIES

By R. GUY PULVERTAFT, F.R.C.S.

DIRECTOR, ACCIDENT AND ORTHOPAEDIC SERVICE, DERBYSHIRE ROYAL
INFIRMARY; ASSOCIATE SURGEON, HARLOW WOOD ORTHOPAEDIC HOSPITAL,
MANSFIELD; CIVILIAN ORTHOPAEDIC SPECIALIST, ROYAL AIR FORCE

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PART I DISEASES OF MUSCLE

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1. PRIMARY MYOSITIS

(1) Dermato-myositis

235] Dermato-myositis is an acute, a subacute or a chronic disease of un- *Pathology*
known aetiology; it occurs, as a rule, during active adult life and is character-
ized by muscular pains, tenderness, weakness and extreme muscular
degeneration and fibrosis. The overlying skin and subcutaneous tissues *Clinical*
become oedematous and hardened, with patches of dermatitis. Severe *picture*
muscular atrophy and contractures eventually occur. The disease may have a
fatal termination within a few weeks, or its course may be prolonged, with
periods of remission; the final outcome, however, is usually fatal. In 50 per
cent of cases, death occurs within 2 years. A goitre is present in many cases.

There is no curative treatment, but orthopaedic measures should be adopted *Treatment*
to prevent or to limit deformities.

(2) Myositis fibrosa

In myositis fibrosa, which is a very rare disease of unknown aetiology, the *Pathology*
affected muscles are gradually replaced by fibrous tissue. The disease usually *Clinical*
commences in childhood or in young adult life, and has a gradual onset. The *picture*
muscles become hard and contracted but, although the skin may become
oedematous, dermatitis does not ensue. Creatinuria is a notable feature in the
acute and subacute forms of generalized myositis fibrosa

There is no treatment, apart from the prevention of deformities.

Treatment

2. GAS GANGRENE

(1) Bacteriology

Gas gangrene is caused by the infection of tissues, particularly of muscle
tissue, by anaerobic organisms of the genus *Clostridium*. There are two main
groups:

(1) Saccharolytic organisms, which grow in carbohydrate media and pro-
duce acid and gas. In this group are *Cl. welchii*, *Cl. oedematiens*, *Cl. tertium*,
Cl. fallax and *Cl. septicum*.

(2) Proteolytic organisms, such as *Cl. histolyticum* and *Cl. sporogenes*,
which grow in meat media and which break down protein into amino acids
and sulphur compounds.

Most infections are mixed, but one or more of these organisms may pre-
dominate. Gas formation is most marked in infections by *Cl. welchii*;
oedema occurs in *Cl. oedematiens* infections, and autolysis in *Cl. histolyticum*
infections. Organisms of the saccharolytic group are usually the first to
appear, and they prepare the ground for the proteolytic organisms to develop.
Anaerobic infections are often associated with the presence of aerobic organ-
isms—chiefly the streptococcus and *Bacillus proteus*—which, by their oxygen-
consuming property, help to produce the necessary conditions for anaerobic
growth.

The main habitat of anaerobic organisms is the soil, but their original
source is animal faeces; they are the normal inhabitants of human and animal
intestines. It follows that cultivated soils are comparatively rich in all organ-
isms, whereas non-cultivated or sandy soil is relatively free. The ready
tendency to form spores makes it possible for the clostridia to remain
alive in soil or in human tissues for long periods.

(2) Pathology

The organisms may be present in a wound without causing disease, and it is recognized that contamination is common, although clinical infection is rare. If conditions are suitable, they may produce a rapidly fulminating disease, or they may lie dormant as spores until further injury or operation causes the infection to become active.

Certain conditions are necessary for the development of clinical gas gangrene; these are contamination of wounds, the presence of dead tissues and anaerobic conditions. Ogilvie (1944) points out that deficient oxygenation may arise from loss of blood, injury to a main vessel, wound tension, or the presence of aerobic organisms, especially streptococci.

In a typical, severe infection, oedema is present within 18–24 hours after the wounding, bullae appear in the skin, and the tissues become distended by blood-stained fluid and bubbles of gas. The vessels thrombose and muscle death occurs. There is little cellular reaction; the disease spreads rapidly throughout the muscle group involved and beyond it, causing massive gangrene. As a terminal event, the organisms finally pass into the blood stream, by which they become widely distributed.

*Mortality
rate*

The mortality rate is high. Ogilvie (1944) records a mortality rate of 50 per cent in a series of 164 cases which occurred in the Middle East in the years 1940–42 inclusive. MacLennan (1946) points out that the mortality rate has been reduced in later series of cases to below 30 per cent, but states that this figure remains fairly constant.

(3) Clinical picture

The clinical course of the disease is marked by severe local pain and swelling, a red-brown serous discharge, progressive toxæmia and, finally, the presence of foul-smelling gas and crepitations in the tissue. Pyrexia is not usually marked, but the toxæmia leads to a rapid pulse rate, a typical grey appearance of the face and an anxious and alert mental state.

*Nature of
the toxæmia*

It is not certain to what extent the responsibility for the toxæmia can be divided between the bacterial toxins and the results of tissue breakdown. It has always been recognized that the influence of polyvalent gas gangrene antitoxin is limited, and the explanation may lie in the possibility that the main source of the toxæmia is the muscle necrosis. Jeffery and Thomson (1944) drew attention to the fact that in some fatal cases death occurred after the arrest of the gangrenous process and that kidney changes, of the kind found in the crush syndrome, were present in these cases. They suggested that the toxic effects may have arisen from the retained degenerated and autolysed muscle.

Macfarlane (1945), reviewing a series of 185 cases which occurred in the

raises the same issue and questions whether the 30 per cent mortality rate may not be due essentially to toxins arising from tissue breakdown.

(4) Treatment

(a) Prophylactic methods

It is generally agreed that despite the use of serum and the sulphonamides and penicillin, the importance of good primary surgery has not been lessened.

Gas infection is to be feared in all lacerated wounds, particularly when muscle is involved, but it may also occur after trivial injuries.

Adequate excision of the wound is essential, with removal of all damaged and ischaemic muscle, the relief of tension by division of fascia, and the removal of foreign material likely to contain organisms. Measures which are likely to diminish the blood supply or to cause tension in the wound must not be employed, such as prolonged use of a tourniquet, tight packing or the application of a tight plaster. *Wound excision*

The question of closure of a lacerated wound or of an open fracture involving severe soft-tissue damage is not one to be settled by a dogmatic statement. In civilian practice many wounds have primary suture performed with excellent results, but in cases of marked severity and in battle wounds it is a wiser policy to leave the wound open and to undertake secondary suture or skin grafting as soon as the wound condition permits. *Closure of wounds*

Ogilvie (1944) points out that in cases in which blood loss has been compensated by transfusion, and primary surgery has been prompt and adequate, gas gangrene is very rare.

Polyvalent gas gangrene antitoxin in a dosage of 22,500 units should be given as a routine in severe wounds in which muscle is involved. Penicillin will be administered as a routine measure against infection by streptococci and staphylococci, but there is considerable evidence to show that penicillin used prophylactically does not influence the development of gas gangrene (Cutler and Sandusky, 1944), despite the fact that the clostridia are penicillin-sensitive. *Chemotherapy*

(b) Treatment of clinical infection

Should it be suspected that gas infection has occurred, it is necessary to undertake prompt surgical measures, combined with the administration of antitoxin, sulphonamides and penicillin.

Polyvalent serum, 60,000–100,000 units, should be given intravenously and the dose should be repeated at intervals of 4–6 hours. Sulphathiazole, 3 grammes, should be given by the intravenous route, and should be followed by oral administration at a dosage level of 10–15 grammes per day. The usual precautions against toxic effects must be taken; alkalis should be given, and the fluid intake kept high. Penicillin, although of uncertain value in preventing the onset of clostridial infection, is of value in limiting the spread of the disease when administration of this drug is combined with surgery, penicillin should be given in doses of 30,000–50,000 units three-hourly.

Continuous intravenous administration of blood or plasma may be required in a severe infection.

The surgical attack should aim at the complete removal of all infected tissue. This may be rendered possible by means of a local excision or by the removal of a muscle group. Loss of a limb may often be avoided, but amputation must not be delayed if more conservative measures do not appear to be sufficient. The amputation may necessarily be of the guillotine type, but it is better to form flaps which may be sutured back to the limb until the stump is free from infection and is safe to cover. *Surgical methods*

When the infection occurs in the hip or the shoulder and amputation is impossible, the wound should be laid fully open and the fascia and muscle sheaths incised to relieve tension.

(c) *Deep x-ray irradiation*

Deep x-ray irradiation has been used for many years in the treatment of gas gangrene, particularly in the United States of America, and in Australia, but it is difficult to assess its value in the routine treatment of these infections. Enthusiastic reports have appeared, but it is not a method that is widely used in Great Britain, and readers should refer to the published papers on the subject. (Smithers, 1941.)

3. TRICHINIASIS

Trichiniasis is an infection by the nematode worm *Trichinella spiralis*. The adult worm (male, 1.4×0.04 millimetres; female, 3×0.6 millimetres) lives for a few weeks only and inhabits the intestinal mucosa of the host. There the female becomes fertilized and at once migrates through the intestinal wall to enter the lymph stream, producing its young in the mesenteric glands. The larvae ($100 \times 6\mu$) are carried by the lymph stream and the blood stream to their resting place in the muscles and become encapsulated there. The larvae may remain encysted and alive for many years, but may die and become calcified.

Pigs and rats are the natural hosts, but man may become a host by eating infected pork.

(i) *Clinical picture.*—The symptoms vary according to the stage of infection. For the first week, during the invasion period, gastro-intestinal symptoms are predominant. Nausea, vomiting and diarrhoea occur, and sometimes blood and mucus appear in the faeces. The symptoms may suggest food poisoning or dysentery. The migratory period, lasting for 7–21 days, is characterized by muscle pains and tenderness which are particularly noticeable in the tongue, laryngeal and intercostal muscles; this period is usually marked by a fever of typhoid form. The blood changes are consistent, showing a high leucocytosis and marked eosinophilia, and help to distinguish the disease from enteric fever and from acute rheumatism.

The stage of encystment is also accompanied by muscle pain, and often oedema and urticaria occur. Cachexia is caused by the absorption of toxins, and death may result from toxæmia.

(ii) *Diagnosis.*—The diagnosis can be established by intradermal injection of antigen or by the flocculation test of Suessenguth and Kline (1944). Biopsy of affected muscle, particularly at the musculo-tendinous insertion, may reveal the encysted larvae and, at a later stage, radiological examination may demonstrate the calcified larvae.

(iii) *Prognosis.*—The prognosis depends upon the severity of the infection; the average mortality rate is about 3–5 per cent, but may rise to 25 per cent in severe epidemics.

(iv) *Treatment.*—There is no specific treatment, and the only safeguard is to ensure that pig meat is adequately cooked. Purgation is advised during the early stages of an infection. Fortunately, the disease is rare in the United Kingdom; this is due to the fact that there is little pig infection and also to the hygienic habits of the population.

Invasion
period

Migratory
period

Stage of
encystment

Mortality
rate

4. OSSIFYING CONDITIONS OF MUSCLE

(1) Traumatic myositis ossificans

When periosteum is raised from bone, ossification occurs in the subperiosteal *Pathology* haematoma. This is a natural process of healing and is of no clinical significance unless it occurs in the neighbourhood of joints and so limits movements, or unless the ossification is very bulky and interferes with muscle action, as on the anterior surface of the thigh.

Injuries in the region of the elbow joint, particularly in children, may be complicated by the development of ossification and function may be severely limited. Early reduction, gentle handling and the avoidance of passive stretching are principles to be rigidly applied, if this condition is to be avoided.

When the ossification has occurred, rest must be enforced until the active *Treatment* phase is over; treatment is then directed towards the gradual restoration of active movements. Considerable re-absorption of the bone may occur, but at times it is necessary to remove consolidated bone in an effort to restore movement.

(2) Generalized myositis ossificans

This is a rare inflammatory condition of muscle, which is followed by wide- *Pathology* spread fibrosis and by the formation of bone in muscles, tendons, fasciae and ligaments.

Isolated tender swellings first appear over a wide area, and bone eventually *Clinical* develops. Repeated exacerbations, usually accompanied by pyrexia, occur *picture* until eventually there are gross deformities.

There is no specific treatment; death finally supervenes as a result of inter- *Treatment* current infection.

(3) Heterotopic ossification

In heterotopic ossification there is a local formation of bone in fibrous and *Pathology* tendinous tissues remote from bone structures. The supraspinatus tendon, tendo Achillis, semilunar cartilages and intervertebral discs are typical sites. Bone may develop in bronchial cartilages in bronchiectasis.

The probable explanation is that calcification may occur in tissues when metabolism is decreased, and later, when revascularization occurs, the calcareous deposits become ossified.

5. DISORDERS OF MUSCLE FUNCTION

The aetiology of the muscular dystrophies and other disorders of muscle function is still obscure, but in recent years considerable advance has been made in our knowledge of the biochemical reactions of muscle activity. Creatine is a normal constituent of muscle and creatinine is excreted in the urine at a constant level. The creatine content of urine is insignificant except in children, and to some extent in women, in whom there is a moderate normal excretion level. The activity of muscle is reflected in the changes of creatine metabolism. Pseudo-hypertrophic muscular dystrophy, for instance, is associated with an impairment of the power to retain exogenous creatine, with the result that the creatine content of the muscle falls and creatinuria

*Changes in
creatinine
metabolism*

occurs. The myotonic states are characterized by a high degree of retention of exogenous creatine and the absence of creatinuria, even in children.

The alterations in creatine metabolism are an indication of the muscle state, but do not tell us why the abnormal condition is present. Investigations into the causation and treatment of *myasthenia gravis* have led to some interesting findings regarding muscle activity. It is not possible to discuss at any length the detailed work which has been performed, but a general outline of the present conclusions can be stated.

It has been demonstrated that acetylcholine is liberated at the myo-neural junction as the result of nerve impulses (Dale, Feldberg and Vogt, 1936), and it is thought that this substance may activate the muscle. The action of acetylcholine is controlled by cholinesterase which breaks down acetylcholine into choline and acetic acid, and the normal function of muscle may depend upon the balance attained between these two substances.

Chemotherapy

The inherent fault in myotonia appears to be an excessive concentration of acetylcholine, whereas the opposite state occurs in myasthenia gravis—a disease characterized by muscle weakness and a low fatigue level. Prostigmin has a rapidly beneficial effect upon myasthenia gravis but increases the muscular spasms in myotonic conditions, and is thought to act by inhibition of cholinesterase. Quinine has the opposite clinical effect, relieving muscle spasms in myotonia but increasing the weakness in myasthenia.

*Removal of
thymus gland*

The overriding control of the biochemical balance at the myo-neural junction is not determined, but it appears reasonable to believe that it may be an endocrine influence. In 1912, Sauerbruck performed a partial removal of the thymus gland in a case of myasthenia gravis, the result being a clinical improvement in the muscle state. In recent years Blalock and his colleagues (1941) in Baltimore, and Keynes (1946a) in London, have produced series of cases in which clinical improvement occurred after complete removal of the thymus gland. Despite the fact that it is uncommon to detect gross changes in the gland that is removed from the myasthenic patient, it does appear that the thymus gland is, in some unknown manner, a responsible factor in the control of the biochemical changes which are involved in muscle activity. It follows, therefore, that some, at least, of the muscle disorders may result from endocrine dysfunction and that they are not true muscle diseases.

*Endocrine
dysfunction*

(1) Muscular dystrophies

(a) *Pseudo-hypertrophic muscular dystrophy*

Pathology

In this condition there is a primary degeneration of muscle, combined with an increase of fat in the connective tissue, which gives the false appearance of muscle hypertrophy. The muscle fibres become swollen and subsequently atrophic and are replaced by fibrous tissue and fat. The disease makes its onset known in early childhood, and the child is becoming increasingly waddling. There is a tendency to lag, and the child is unable to rise by rolling over on his face, rising on hands and knees and climbing up to an erect position by the help of his hands.

*Clinical
picture*

In a well-advanced case the calves are enlarged, and there is weakness of calf, quadriceps and gluteal muscles in particular. The spinal muscles are involved, producing a characteristic lordosis. In the upper limbs, the shoulder-girdle

musculature is affected and ultimately most of the trunk and limb muscles become involved, with final contractions and spinal deformity. Death usually occurs in adolescence, the cause being intercurrent infection.

(b) *Primary atrophic types of muscular dystrophy*

These various types of muscular dystrophy show the same pathology, but the apparent hypertrophy is absent. Classification is made according to the regions involved, but mixed types are occasionally seen. They include the facio-scapulo-humeral type (Landouzy-Déjerine), facio-scapular or juvenile type (Erb) and the distal type affecting the forearms and legs (Duchenne-Aran).

Examination of the urine gives the best indication of the state of the patient. *Biochemical examination* As the disease progresses, the characteristic findings are (i) an increasing degree of creatinuria, (ii) a progressive fall in urinary creatinine, and (iii) a low creatine tolerance to test doses given orally.

There is no satisfactory treatment for the muscular dystrophies. Glycine, 10 grammes, given daily, has been claimed to produce improvement, but it has at the best only a temporary effect. Prostigmin, for which hopes were entertained in view of its action on myasthenia gravis, has proved disappointing.

(2) Amyotonia congenita (myotonia congenita: Oppenheim's disease)

Amyotonia congenita is a rare familial and probably hereditary disease which may be present at birth or which may appear during the first year of life.

The muscle fibres are very small but occasionally giant forms are seen. The *Clinical picture* muscles are small and weak, and marked flaccidity is present. Distribution is symmetrical and affects the legs, trunk and arms but not the face.

Significant biochemical findings are (a) low excretion of creatinine, (b) increased excretion of creatine, and also increased excretion of exogenous *Biochemical findings* creatine; the increase, however, is not so marked as it is in progressive muscular atrophy.

Death may occur in severe cases, but in mild cases there is a tendency for slow improvement to occur, and occasionally an almost complete recovery is seen. There is no adequate treatment, but physical treatment on general lines *Treatment* is of some benefit.

(3) Myotonia atrophica

This rare familial hereditary disease is characterized by defective relaxation *Clinical picture* of muscles and weakness; the onset occurs during the third or fourth decade of life. The general health is not affected, but eventually, after many years, death occurs from intercurrent infection.

The disease affects the facial muscles, the forearm and hand, and the leg muscles. The myotonia is the remarkable feature; voluntary contraction of muscle is followed by a delayed relaxation.

The aetiology is unknown and there is no curative treatment. Quinine abolishes the myotonia, but has no effect upon muscular power. The *Biochemical findings* chemical findings are creatinuria and a high excretion of a test dose of creatine.

(4) Myotonia congenita (Thomsen's disease)*Biochemical findings*

Myotonia congenita is a familial and hereditary disease of unknown aetiology, with an onset in early childhood. There is widespread muscle hypertrophy, associated with spasms, which occur at the beginning of muscular action; these spasms have a duration of 5-10 seconds and are followed by slow relaxation. The progress of the disease is slow, and there is a tendency for remissions to occur. The general health is not affected, and deformities do not occur. Creatinuria is absent and there is an abnormally high retention of a test dose of creatine.

Hypothyroidism may be associated with the disease, and thyroid extract may be of value during infancy and childhood. Quinine has the power of abolishing the myotonia but only for a limited period.

(5) Myasthenia gravis*Clinical picture*

This uncommon disease is characterized by abnormal and often extreme muscle weakness. The condition has an insidious onset and is generalized in its distribution, but there is a characteristic involvement of the ocular muscles. Remissions for long periods frequently occur, but the ultimate prognosis is poor. Occasionally the disease follows an acute course, leading to death in a few months. The average age of onset is early adult life, but symptoms may arise in the very young and after middle age.

Pathology

The aetiology is uncertain, but much knowledge has been gained in recent years. Frequently, an enlargement of the thymus gland has been noted, and radiological examination of the chest in all cases of the disease is recommended. It is believed that there is a lack of acetylcholine at the myo-neural junctions and that this is due to an increased formation of inhibitory substance by the thymus gland. Successful results have been obtained by the removal of the thymus gland (Blalock and his colleagues, 1941; Keynes, 1946b), but the results are somewhat uncertain. The administration of Prostigmin by mouth (Remen, 1932) is generally considered to be the basic treatment for the majority of cases, and by careful administration it is possible to maintain an improved condition for long periods.

Treatment

It is not possible to enter into a full discussion of this interesting disease, and recent works should be consulted.

(6) Familial periodic paralysis

Familial periodic paralysis is a disease which is characterized by attacks of flaccid paralysis spreading from the legs to the arms, trunk and neck. The

Metabolic dysfunction

The aetiology is unknown and there are no definite pathological changes. It has been suggested that there is a metabolic dysfunction, and it has been noted that serum potassium diminishes during an attack. The attacks may be cut short by the administration of potassium salts.

6. TUMOURS OF MUSCLE

All the

(1)

angioma and sarcoma.

(2) Arising from the muscle fibres are the true muscle tumours—rhabdomyoma and myoblastoma of striated muscle and leiomyoma of unstriated muscle.

(3) Secondary deposits of carcinoma or sarcoma.

(1) Tumours of associated tissue

(a) *Fibroma*

This tumour is typically benign, but the "desmoid tumour" of the anterior abdominal wall infiltrates widely and tends to recur locally after removal. Ninety per cent of the desmoid tumours occur in women, and are usually found in women who have borne children.

(b) *Haemangioma*

The haemangioma is of importance because of the severe haemorrhage which is likely to occur if the surgeon does not suspect the nature of the tumour before operation. It may be capillary, arterial, venous or cavernous in type. It may be present at birth and is usually found before the age of 30. The tumour may extend to involve nearly all the structures of the limb, including the bone, and the limb becomes greatly enlarged.

Growth may continue for many years, and complications such as infection, thrombosis and haemorrhage may occur. Sarcomatous changes with metastases are occasionally seen. Radiotherapy is suitable for the capillary cavernous haemangioma, but not for the arterial and venous types. *Radiotherapy*

(c) *Lymphangioma*

A lymphangioma may form part of a haemangioma and may be the predominant feature, but Ewing claims that a pure lymphangioma of muscle has not been described. (Ewing, 1940.)

(d) *Sarcoma*

Connective tissue, nerve tissue and fat may give rise to sarcoma. The neurosarcoma may extend along the nerve trunk and produce apparently separate tumours higher in the limb. The prognosis of these tumours is bad, and although pseudo-encapsulation may tempt the surgeon to perform enucleation, wide excision or amputation, according to the site of the tumour, is essential. Radiotherapy is of value only in liposarcoma and may be used as a palliative measure or in combination with wide excisions. (Ackerman and Regato, 1947.)

(2) True muscle tumours

The tumours of striated muscle may be divided into two groups: (a) rhabdomyoma or rhabdomyosarcoma, which shows longitudinal and transverse striation; (b) myoblastoma, which shows longitudinal striation only.

(i) *Pathology*.—Tumours in each of these groups may have benign or malignant characteristics. Cappell and Montgomery (1937) consider that the malignancy of these tumours depends upon the existence of primitive round cells or spindle cells which metastasize and is not due to dissemination of the striated fibres and myoblasts. Secondary deposits are most commonly seen in the lungs, but may occur in bone and liver.

(ii) *Clinical picture*.—These tumours may appear at any age; they have been recorded as occurring at ages as early as 2½ years and as late as 70 years. *Age incidence*. The slowly growing forms are encapsulated, but the more malignant types

infiltrate freely. Gordon-Taylor (1940) describes three clinical groups: (a) slow-growing tumours which recur after excision and which cause death within 2-3 years; (b) fulminating growths which lead to death within a year, whatever treatment is given; (c) tumours which, if untreated, remain stationary for some years before undergoing sudden change and rapid growth.

(iii) *Diagnosis*.—The tumours are usually painless and, in the early stages, consist of an isolated swelling which is deep in muscle and is movable when the muscle is relaxed.

It is rarely possible to diagnose a muscle tumour as either simple or malignant; in its early stages, and in cases in which the growth is readily accessible, it should be removed by excision and its nature should be determined by histological examination.

(iv) *Treatment*.—If the tumour is radio-sensitive, excision should be followed by x-ray therapy or by radium implantation. Radium appears to give the better results.

*Radium
implantation*

If the section shows the tumour to be radio-resistant and, in particular, if it is a rhabdomyosarcoma, amputation is the only method of treatment that can offer hope of success.

*Surgical
treatment*

When the neoplasm is deeply placed and inaccessible for removal, a biopsy should still be performed, followed by irradiation or amputation, the choice depending upon the radio-sensitivity of the tumour.

The other true muscle tumour is that arising in unstriated muscle—the leiomyoma. This is a common tumour and is most frequently seen in the uterus. It also occurs in the stomach, bladder and ovary. It is usually benign but occasionally it undergoes sarcomatous changes.

PART II DISEASES OF THE TENDONS AND TENDON SHEATHS

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1. TENO-SYNOVITIS

(1) Traumatic or crepitating teno-synovitis

A low-grade aseptic inflammation may develop in a tendon or muscle sheath after an isolated injury, or after the repeated trauma of over-use.

Inflammatory changes occur on the surface of the tendon and sheath or the paratenon, and may extend into the muscle. Howard (1941) has demonstrated that deposits of fibrin in the oedematous tissues are responsible for the characteristic silky crepitus. Pathology

The condition is most commonly seen in the extensors of the thumb and in the radial extensors of the wrist in the lower forearm, but may also be seen in the flexors of the fingers, the long head of biceps, the tendons around the ankle joint, and the tendo Achillis. The changes may, in fact, occur in any tendon which is particularly likely to be subjected to over-use or strain.

Treatment consists of rest obtained by means of a type of splintage suitable to the part involved, and an early acute case may be expected to settle down in 10-14 days. In chronic cases, cure is more difficult, and prolonged rest may be required, with a graduated return to activity. Treatment

When the condition has fully settled down, there appears to be little tendency for recurrence, except in certain rare cases in which oxaluria is present. Urine analysis should be performed in all persistent cases.

(2) Infective teno-synovitis

(a) Acute

(i) *Pyogenic teno-synovitis*—Pyogenic or suppurative teno-synovitis is most often seen in the flexor sheaths of the fingers and hand; usually it occurs as a result of direct infection caused either by a prick or by spread from an adjacent focus of infection.

The treatment and prognosis of this serious condition have been radically altered with the introduction of penicillin; the present approach to the problem is fully discussed by Barron (1948).

Owing to the greatly improved results of treatment, cases in which the infection has been successfully overcome are now more often seen, but the inflammatory reaction is followed by the formation of adhesions which limit the tendon range. A late tenolysis may do much to restore function in these cases. Limitation of tendon range

(ii) *Gonococcal teno-synovitis*.—This condition results from a blood-stream infection and, although it may occur at any stage of the disease, it is usually seen in the early weeks of a neglected infection. The incidence of gonococcal teno-synovitis has decreased as a result of sulphonamide and penicillin therapy. The condition may vary from a serous reaction to a low-grade inflammation, but only rarely does true suppuration occur. Treatment is directed on general lines.

(b) Chronic

(i) *Syphilis*.—A gummatous teno-synovitis is very rarely seen but, when present, may lead to confusion with tuberculous disease.

Either a local or a diffuse gummatous formation may occur and, if neglected, will give rise to a syphilitic ulcer. A positive Wassermann test and the rapid response to anti-syphilitic treatment will establish the diagnosis.

(ii) *Tuberculosis*.—Chronic teno-synovitis is almost always due to tuberculosis. It may arise as a result of direct local infection; occasionally, however, the cause is an infection carried by the blood stream.

The commonest situation is the flexor sheaths of the finger or thumb, but the extensor sheaths of the hand, and the tendon sheaths of the ankle and foot, may be the site of the disease.

It is occasionally seen in children but most often in male adults, particularly in those who work with cows, for example, farm hands, fellmongers or butchers, and may be the only manifestation of the disease in the body.

Pathology.—The disease affects the synovial membrane and may exist for several years before the tendons become seriously involved. A serous exudate is formed, causing distension of the sheath; the sheath gradually thickens and fibrin formation occurs, producing the typical melon-seed or rice bodies.

The sheath becomes progressively involved until it is replaced by a *granulomatous mass throughout its whole extent. Eventually caseation occurs and a sinus is formed.*

Clinical picture.—Diagnosis is not difficult when the disease is well advanced, but the condition may be overlooked in its early stage by both patient and physician, because of its gradual and insidious development. The presenting symptom is usually a painless swelling along the course of a tendon, and when a restraining ligament exists, an hour-glass appearance is produced. When the ulnar bursa is affected, a typical filling of the palm and wrist is present, with communication deep to the transverse carpal ligament, giving rise to the condition known as compound palmar ganglion. In well-established cases the typical creaking feel of melon-seed bodies can readily be detected.

There is but little pain and tenderness, and the joint range and tendon action are not materially affected until late in the disease.

Compound
palmar
ganglion

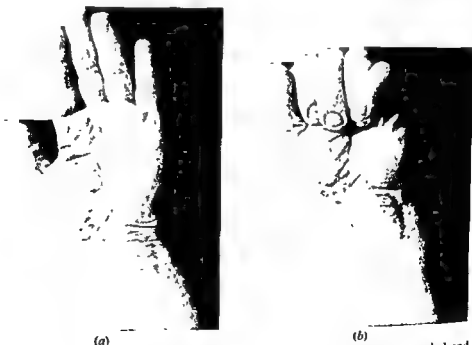


FIG. 81 (a) and (b)—Result obtained after complete removal of tuberculous radial and ulnar bursae.

Treatment.—A general examination of the patient should be made in order to find any other tuberculous focus; treatment for the general condition should be directed along standard lines.

Treatment of the local disease should be both conservative and radical. Splintage alone is unsuccessful, and can be applied for many months or even for years without effective result. Complete surgical extirpation of the affected sheath is the only satisfactory procedure but, in an active case, it is often wise to combine general treatment in a sanatorium with local treatment in the form of splintage, for a few months preceding operation.

*Surgical
extirpation
necessary*

The excision of the sheath is a tedious and time-consuming procedure and should always be performed in a bloodless field. A full exposure with carefully planned skin incisions should be used, and the entire sheath must be removed.

The results obtained after complete removal are very satisfactory and it is remarkable how little the tendon action is affected (Figs. 81 and 87).

*Results of
treatment*

2. TENOVAGINITIS STENOSANS

This interesting condition is seen in two distinct groups of cases: in infants and young children, in whom the site is typically the flexor pollicis longus at the level of the metacarpo-phalangeal joint, and in adults—usually women—in whom the fingers at the same level are most commonly affected.

Both the sheath and the tendon are involved. The sheath is thickened and an annular constriction is produced; the tendon shows a corresponding enlargement proximal to the isthmus of the sheath. The presenting symptom in children is commonly a fixed flexion contracture of the terminal interphalangeal joint, which can be mistaken for either a dislocation or a true joint contraction. In adults, a typical trigger-finger syndrome is usually seen, and the digit can be straightened with a palpable click. The thickening in the sheath is sometimes mistaken for an abnormal sesamoid.

Pathology

*Clinical
picture*

The treatment is operative. A small transverse incision is made in the skin crease, and the sheath is exposed and opened in the line of the tendon. In children it is unnecessary to do more than to open the sheath and allow a free run of the tendon, but, in adults, it is advisable to remove a section of the annular constriction.

Treatment

3. DE QUERVAIN'S SYNDROME

In 1895 de Quervain described a condition of narrowing of the fibrous sheaths of the abductor pollicis longus and the extensor pollicis brevis, as they pass along the groove on the lateral surface of the lower end of the radius, and he suggested that the condition arose from chronic irritation. There is a local tenderness, and sometimes a visible swelling is present over the lateral aspect of the radius. Movements of the thumb cause pain, and the diagnosis is confirmed by the acute pain produced when the thumb is forcibly flexed while the wrist is held in ulnar deviation. The only satisfactory treatment is complete longitudinal opening and partial excision of the sheath. The results of treatment are uniformly good.

*Clinical
picture*

Treatment

4. TUMOURS OF TENDON SHEATHS

(1) Benign tumours

The benign tumours are chondroma, osteochondroma, myochondroma, lipoma and fibroma; all are comparatively uncommon.

(2) Malignant tumours

(a) Sarcoma

The malignant tumours are sarcoma and synovioma. Usually sarcoma is highly malignant; the tumour metastasizes widely, but in occasional cases the course is one of merely local recurrence over many years.

(b) Synovioma

Metastases

The synovioma is also very malignant and gives rise to extensive metastases. It is usually seen in early adult life, the common sites of origin being the knee, ankle, foot, elbow, wrist or hand. These tumours are usually well defined in their early stages and may pass through a latent period before local and distant invasion occurs. Not infrequently they penetrate bone and may be confused with a primary bone tumour.

Microscopically, the synovioma shows a large number of closely arranged spheroidal or spindle cells with a mucoid interstitial tissue; it may also show synovial spaces. Mitotic figures are common and blood-vessel invasion is apt to be free.

Treatment

Treatment consists of efficient local excision; if the tumour is extensive, however, and cannot be safely removed without mutilation, primary amputation should be performed.

(3) Giant-cell tumours

The xanthomatous giant-cell tumour is in a separate class because there is some doubt whether it is a true neoplasm. It is a fairly common tumour, occurring especially in the tendon sheaths and the aponeuroses of the hands and feet. It is encapsulated, firm and yellowish in colour. Metastases do not occur and local recurrence is rare. Histological examination shows a multiplicity of cell forms, including giant cells of the foreign-body type, xanthoma cells containing cholesterol, cartilage cells and fibroblasts. The so-called foam cells are an artificial product of section preparation during which the cholesterol is dissolved out of the xanthoma cells, leaving empty vacuoles.

Radiotherapy

Radiotherapy is effective in these cases, as in the other xanthomatous lesions.

(4) Ganglion

The aetiology of the condition is still in doubt, and it is not certain whether the ganglion should be grouped with the neoplasms.

These are common cystic tumours, occurring in the region of the wrist, hand, ankle and foot and occasionally in other parts. The ganglion is composed of cystic spaces which are filled with clear greyish jelly and lined with connective tissue. It does not have a true endothelial lining, though this may be simulated by the fibroblasts. The ganglion is attached by a fibrous band to the tendon sheath or the joint capsule, and usually does not have direct communication with either.

Excision

It is often possible to disperse a ganglion by means of firm pressure but, if it is not possible to do so, excision is the only method.

PART III

TRAUMATIC LESIONS OF MUSCLE AND TENDON

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1. RUPTURE OF MUSCLE AND TENDON

(1) Spinal muscles

Concentrated research into the causes of low backache and sciatica in recent years, and the confident diagnosis of prolapsed disc with its successful treatment, have shown that many cases which were previously diagnosed as muscle strain and fibrositis are due to the disc syndrome.

Nevertheless, the strained back still occurs as a very real clinical entity. It may result from any action which produces a violent muscular contraction,

and it is heralded by severe local pain, usually in the lumbar muscle group. In a severe case, agonizing pain results from any movement and the sufferer lies in some position of ease, dreading the next spasm.

(i) *Diagnosis*.—Differential diagnosis from a disc lesion is not always simple. The spine may be held rigid by protective muscle spasm and, although true leg pain and neurological signs of root pressure are absent, straight leg raising may be limited and may cause increased pain. The careful localization of a tender area—which is often deep and in proximity to the transverse processes—and the injection of a local anaesthetic will produce considerable and sometimes dramatic relief of pain, allowing the patient to move freely and to get up without fear.

(ii) *Treatment*.—Treatment is designed to reduce subsequent adhesion formation and deep contractures. Early active movements are prescribed, and, if necessary, are assisted during the painful stages by injections of Novocain. Diathermy and massage should be used to disperse the deep haematoma.

As in other muscle and tendon strains, recurrence is not uncommon and may follow comparatively minor strains or over-use. Many of these cases occur in workmen who are receiving compensation and, on account of the paucity of physical signs in a chronic case, unjust accusations of malingering are sometimes made.

The recurring and chronic cases should be treated by deep heat, massage, exercises and manipulation under anaesthesia, but in persistent cases it should be recognized that a change of occupation may be the only practical solution. Manipulation must be performed only after a full investigation, the purpose of which is to exclude other causes of low backache, particularly the prolapsed disc. Forcible manipulation performed in a case of displaced disc may be harmful, and indeed disastrous, for cord pressure and paraplegia may result from severe displacement of a disc.

*Contra-
indications
to manipula-
tion*

(2) Biceps brachii

Rupture of the long head of biceps in the bicipital groove is usually associated with roughening or adhesions in the groove. Occasionally the muscle belly or the tendinous insertion to the bicipital tuberosity of the radius may rupture.

Treatment.—In the majority of cases, long-head rupture occurs in middle-aged or elderly men, and treatment is unnecessary, since the disability is slight. In a younger subject, repair is advisable and the most practical measure is to suture the ruptured long head to the short head. Muscle-belly ruptures may be repaired by means of direct end-to-end suture; the insertion ruptures may be restored by re-insertion to the bicipital tuberosity, stainless-steel wire and the withdrawal technique of Bunnell (1944) being used.

*The Bunnell
withdrawal
technique*

(3) Triceps

Rupture of the triceps insertion is usually associated with detachment of a flake of bone from the olecranon. As a rule, loss of extension power is not complete, owing to the lateral expansion remaining intact, but marked weakness occurs and there is a palpable gap proximal to the olecranon.

Treatment.—The tendon should be re-attached to the olecranon, a transverse drill hole being made in the bone so as to give a satisfactory hold for

the sutures. Plaster fixation in a mid-position between full extension and right-angle flexion is maintained for 4 weeks, after which graduated exercises are commenced. Normal return of function can be anticipated.

(4) Tennis elbow

Tennis elbow is a familiar and annoying complaint which is easily diagnosed but difficult to cure. The syndrome consists of pain in the forearm and tenderness over the outer aspect of the elbow at the origin of the extensor carpi radialis longus and extensor carpi radialis brevis. The pain is experienced during any action which brings into play the radial extensors of the wrist. A simple test is to ask the sufferer to lift a bucket of water, with the forearm in supination; on rotating the forearm to the pronated position, pain is immediately experienced as the wrist extensors contract.

In the great majority of cases the condition is due to strain or to partial rupture of the muscle origin from the lateral epicondylar ridge, which gives rise to oedema and subsequent adhesions.

Treatment.—The choice of treatment is decided by the stage of the condition when it is first seen. Acute tennis elbow with a history of a few days' duration is best treated by rest, either by a dorsiflexion plaster applied to the wrist or by a simple sling, until symptoms have subsided. Unfortunately, this may take a considerable time—several weeks or even months.

In late or chronic cases, more active measures are required. Manipulation usually produces a dramatic cure in cases in which there is true limitation of elbow extension with the wrist held in extreme flexion. Mills's procedure is to force full extension of the elbow while the forearm is held pronated and the wrist flexed (Mills, 1928). Another method is to force a sudden adduction action at the elbow while the joint is held in the extended position (Cyriax, 1936). These manoeuvres are best done under short anaesthesia and should be followed by immediate active exercises to maintain the full length of the wrist extensors. *Manipulation techniques*

Manipulation is often ineffective in the treatment of cases in which there is no true limitation of extension, and in this group cure is difficult. Diathermy, massage and injection of local anaesthetics are sometimes successful, but if the symptoms persist, an open operation and section and suture of the muscle origin, followed by gradual resumption of activity, will usually produce a cure. *Operative treatment*

(5) Quadriceps

The quadriceps may be ruptured, at its attachment to the patella, by the same mechanism which causes a transverse fracture of the patella. A violent disruptive force is produced when flexion of the knee is combined with sudden strong contraction of the quadriceps, as occurs when an individual stumbles and attempts to save himself. Fracture of the patella is more apt to occur in subjects under middle age, whereas muscle rupture more often occurs in those over middle age, but the generalization is not always true.

The diagnosis is readily made on account of the marked but not complete loss of extension power. A palpable gap can be felt above the patella, although this tends to be obscured by the haematoma. Haemarthrosis occurs if the suprapatellar pouch is torn. *Diagnosis of rupture*

Treatment.—It is possible to obtain a fair result by means of splintage applied in extension which is maintained for 3–4 weeks and is followed by graduated exercises; in the average healthy patient, however, operation should

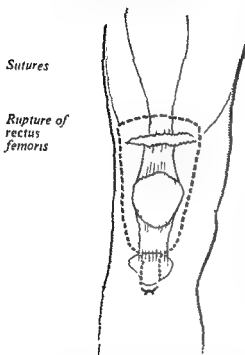


FIG 82 —Dunn's tension stitch used in repair of ruptured quadriceps. The ends of the wire are tied over a gauze pad on the skin surface.

always be undertaken, the muscle being carefully sewn to the patella by strong sutures. There is, in this instance, no inherent advantage in the use of stainless-steel wire; catgut is the more suitable material. The removable wire tension suture advocated by Dunn (1934) may be used with advantage to relieve strain on the suture line (Fig. 82).

Sometimes the rectus femoris alone is ruptured; in this case there is little reaction and extension power is not appreciably weakened, but the alteration in muscle contour makes the diagnosis clear. Treatment depends upon the age of the patient and upon the time that has elapsed since injury occurred. In the patient who still expects to lead an active life, it is advisable to repair an early rupture, but in the elderly patient and in all late cases it is probably wiser to refrain from treatment, since the decrease in function is comparatively slight.

(6) Calf muscles

Partial rupture of the calf muscle is a fairly common injury and is a cause of acute discomfort. Various sites of injury have been described—the heads of the gastrocnemius, the junction of gastrocnemius and soleus and the soleus origin from the tibia. As the syndrome and treatment, however, do not differ, there is no real need for careful distinction.

The injury usually results from a sudden strain during athletic activities. There is pain and acute local tenderness in the muscle belly, but the power to rise on the toes remains.

Treatment.—Treatment should be by injection of local anaesthetic and by active use assisted by physiotherapeutic measures, the aims being to ensure absorption of the haematoma and to avoid the occurrence of fibrosis. The condition usually settles down in a few weeks but there is a tendency towards recurrence.

Rupture of
plantaris
tendon

Rupture of the plantaris tendon is frequently diagnosed, but it is doubtful whether this lesion is a true entity. It has been noted by many observers that in cases in which there is rupture of the tendo Achillis, for which an open repair is performed, the plantaris tendon, if present, is invariably intact. The so-called plantaris rupture is in fact a misdiagnosis for a soleus rupture.

(7) Tendo Achillis

Strain or partial rupture may be caused by a sudden muscular contraction or by repeated subminimal strains during an athletic contest, particularly in long-distance running. It is responsible for causing severe pain and disability. The tendon should be rested by placing felt pads on each side of it and by

rapping the ankle in plantar flexion, combined with raising of the heel. Recovery usually takes 6-8 weeks, but the disability is apt to recur even after the lapse of some years. This condition is often more troublesome than is a complete rupture; the latter usually shows complete recovery after operative repair.

Complete rupture results from a violent muscular contraction such as may occur in jumping, badminton, netball or tennis, or by stepping awkwardly down stairs. The tear is transverse, but as the tendon fibres tend to give way at slightly different levels the tendon ends are usually frayed. This injury is not uncommonly confused with a partial rupture, because the tendon sheath becomes filled by a tense haematoma and the illusion of an intact tendon is produced. Careful examination, however, will always elicit the presence of a gap in both early and late cases, and it can be demonstrated that the apparent power of plantar flexion is produced by *tibialis posticus*, *peronei* and *extensor digitorum longus* and not by true *tendo Achillis* action.

Treatment.—Operative repair should be undertaken in all cases in which there is complete rupture. Recent cases present no difficulty and end-to-end apposition and suture can be readily performed. In late cases a slide elongation is usually required at the musculo-tendinous junction to compensate for secondary muscle shortening, before the tendon ends can be opposed. A clean end-to-end contact is essential for a sound union, and suture should be performed with stainless-steel wire, gauge 34-36, the Bunnell stitch being used (Bunnell, 1944). The circumference of the union should be completed with a few simple sutures (Fig. 83). When the *plantaris* is present it may be used with advantage to strengthen the union by detaching the distal end and interlacing it through the *tendo Achillis* above and below the union as a living graft.

Occasionally neglected cases are seen in which the final elongation and suture appears attenuated and unconvincing, owing to severe muscle shortening. The union in those cases may be strengthened by transfer of the distal ends of *tibialis posticus* and *peroneus longus* to the lower fragment of the *tendo Achillis* or direct to the calcaneus.

Post-operative treatment consists in plaster fixation in equinus for 6 weeks, the equinus being gradually decreased by means of changes of plaster. This is followed by the wearing of a raised heel for a further 4 weeks, the height of the heel being gradually reduced to normal during this period.

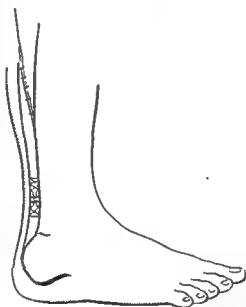


FIG. 83—A combined slide elongation and suture in a late case of *tendo Achillis* rupture.

Recurrences

Post-operative treatment

2. DISLOCATION OF TENDONS

(1) *Biceps brachii**Physical signs*

The biceps tendon may dislocate from the bicipital groove as the result of rupture or because of laxity of the transverse humeral ligament. Pain and a sensation of insecurity are experienced. There are few physical signs; there may be tenderness along the line of the tendon, and, occasionally, in thin subjects, the tendon can be felt to slip. Gilcreest (1936) describes a method of determining the presence of a slipping tendon. With a weight held in each hand, the extended arms are brought to the overhead position, maintaining external rotation. The arms are then lowered to the side in the coronal plane. At approximately 110–90° abduction, a snap can be heard and felt on the abnormal side, and is accompanied by pain in the shoulder and upper arm.

Treatment.—Gilcreest recommends a technique in which the long head of biceps is divided and joined to the short head at the coracoid insertion.

(2) *Gluteus maximus*

F. Wood Jones (1920) has discussed the occasional presence of a tendinous band on the deep surface of the gluteus maximus. On active flexion of the hip while it is internally rotated, this band may slip over the great trochanter with an audible snap. Once the condition has occurred it persists, and may cause considerable annoyance and inconvenience.

Treatment.—Treatment consists of division of the band, which is sutured back upon itself behind the great trochanter. In one of the writer's cases a cure resulted from a partial removal of an unduly prominent trochanter.

(3) *Biceps femoris*

The biceps tendon has been known to slip over the head of the fibula when the tendon is attached more distally than is normally the case. McMurray (1937) recommends that the tendon attachment should be extended proximally on to the fibular head by the method of rawing the bone and sewing down the tendon by deep suture.

The common peroneal nerve must, of course, be identified and retracted.

(4) *Semitendinosus*

This tendon curves behind the internal femoral condyle; forward displacement of the tendon is prevented by the deep fascia. If the fascia is torn or stretched, the tendon may slip over the condyle as the knee is fully extended, thus causing pain and a feeling of insecurity.

Division of tendon

Treatment.—Treatment is not always required but, should the condition be disabling, the most practical measure is to divide the tendon. The subsequent loss of power is barely appreciable (McMurray, 1937).

(5) *Peroneal tendons*

The peroneal tendons run together behind the external malleolus in a groove which is converted into a canal by the superior peroneal retinaculum. If this groove is abnormally shallow, or if the retinaculum is lax or torn, the tendon may dislocate forwards on to the lateral subcutaneous surface of the malleolus, when the foot is dorsiflexed. Pain and insecurity are experienced.

Treatment.—Occasionally an acute injury may be treated successfully by means of firm strapping and the wearing of a shoe-heel which is flared and

wedged on the outer side, but, more usually, operative treatment is required. Several operations have been described; of these, two may be mentioned.

Operative techniques

(1) An osteo-periosteal flap is raised from the subcutaneous surface of the external malleolus and is hinged posteriorly, to be sutured to the astragalus. Plaster fixation for 4 weeks and, afterwards, graduated exercises comprise the after-treatment (Jones and Lovett, 1929).

(2) A strip of tendon is dissected downwards from the tendo Achillis, leaving it attached at its calcanean insertion. This strip is passed through drill holes in the external malleolus and is sutured back upon itself. After-treatment is given as described above (E. Jones, 1932).

3. TENDON INJURIES IN THE HAND

(1) General discussion

Tendon injuries in the hand are common occurrences and they can lead to severe disability. All injured hands should be assessed by an experienced surgeon and should not be treated in the casualty department without adequate supervision. It is essential to reach an early decision as to whether a digit can be saved or whether it would be better to amputate without delay. Treatment should be conservative, particularly in regard to the thumb, but in severe crush injuries of a single finger involving loss of skin, division of tendon and bone and joint damage, it is not always possible to restore function. In such cases, much invalidism and stiffness of the remaining fingers may be avoided if an early amputation is performed.

Careful assessment of injury

The problem of late repair must also be approached in a critical spirit. It is useless to attempt secondary tendon reconstruction in the presence of severe scarring, or of trophic changes resulting from digital nerve damage, or under the handicap of a stiff joint. These complications must receive attention before tendon surgery is undertaken, since, otherwise, the results will be consistently disappointing.

(2) Physiology of tendon repair

Tendons passing over joints which can be flexed through an angle are enclosed in synovial sheaths, but in those which slide in constant straight lines or merely straighten joints from the flexed position, these sheaths are not present; in place of the sheath there is a loose elastic tissue which is termed the paratenon.

When a tendon is divided within a synovial sheath, considerable separation occurs, the ends become rounded and there is little or no attempt on the part of Nature to effect union. The tendon which is severed in paratenon separates to a lesser extent, and Nature attempts to heal the breach by proliferation from the tendon ends.

In certain parts of the hand the tendons are held from retraction by their anatomical arrangement; correct splintage will permit sound union and restoration of function to occur, without surgical interference. This is illustrated by division of the extensor tendon over the distal or proximal interphalangeal joint, the metacarpo-phalangeal joint or the back of the hand.

The healing of tendons which are held in apposition either by external splint-
age or by internal suture takes place in an orderly and progressive fashion.

Process of repair

During the first week the tendon ends become glued together by a fibroblastic reaction from the tendon sheath or the paratenon. In the second week tendon cells grow out from the tendon ends into this mass and the gap becomes bridged by tendon fibres. During the third week the union becomes firmer and the separation between the tendon junction and the surrounding tissues commences. After the third week resolution sets in and the general reaction subsides until the union eventually takes on the appearance of normal tendon.

It is clear, therefore, that no reliance can be placed on the occurrence of tendon union until well on in the fourth week. It follows that, if early movements are to be encouraged, both the technique of suture and the material used must be faultless, since the strain falls directly upon the suture material.

(3) General technique

Individual tendon injuries will be discussed in detail later, but there are certain principles which should guide those who are called upon to perform tendon surgery in the hand.

Precision in technique

All human tissues should be handled with care, and this rule applies particularly to the hand. Bunnell (1944) uses the phrase "atraumatic surgery" and this is the first essential principle for successful surgery of the hand. The most delicate instruments—similar to those employed in ophthalmic surgery—are used; all crushing of tissues and unnecessary interference with gliding surfaces are avoided and the minimum of foreign material is left in the wound.

Application of tourniquet

Fibrosis and cicatricial contracture—the enemies of function—must be avoided as far as possible, and healing should be obtained with the minimum

each 1½-hour period. The tourniquet is released before wound closure is undertaken, and a hot saline swab is applied while the limb is elevated for a few moments. The tourniquet is finally applied again, the wound is closed and a firm compression effect is produced by the application of wool and a crêpe bandage before the blood is permitted to re-enter the limb.

Penicillin

It is a wise precaution to give a short course of penicillin therapy; this is not because the hand is apt to become septic—for indeed it is well protected by its great vascularity—but because sepsis can cause irreparable damage, and even minor degrees of infection will delay healing and result in fibrosis.

(a) Sutures

Steel-wire technique

(i) *Suture material.*—Stainless-steel wire is the most suitable material that is at present available for tendon suture. It causes little or no reaction and, provided that kinking is avoided, it is sufficiently strong to permit graduated active movements before physiological union has occurred. The wire should be obtained in 18-inch lengths, with a small, straight, cutting needle attached

which is passed through a needle-eye in the usual fashion and drawn through tendon will often tear the tendon fibres apart. A reef knot should be used, and it is advisable to cut the two ends of the wire separately so as to avoid the danger of opening the knot.

Bunnell (1944) advises the use of wire of 34-35 gauge, and he employs a removable suture. Alternatively, 40-gauge wire may be used; it may be left in the tendon indefinitely, without giving rise to reaction and fibrosis.

(ii) *Types of suture.*—The types of suture which may be employed are as follows.

(1) The Bunnell end-to-end stitch (Fig. 84 (a)). This is the standard stitch which is used for joining two tendons of approximately equal diameter.

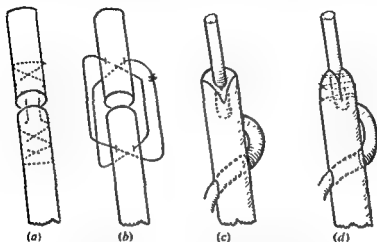


FIG. 84.—(a) Bunnell end-to-end stitch; (b) double right-angle stitch, (c) and (d) stages in forming interlacing stitch.

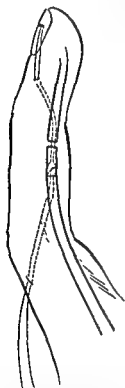


FIG. 85.—The Bunnell removable stitch. Note proximal wire loop for pulling out stitch.

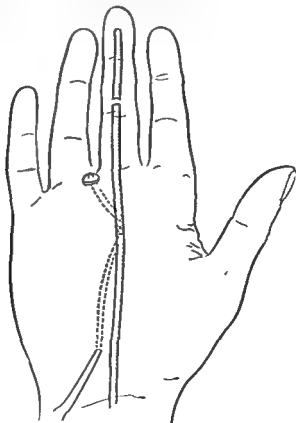


FIG. 86.—The Bunnell stitch used for "suture at a distance". Note proximal wire loop for pulling out stitch.

(2) The double right-angle stitch (Fig. 84 (b)) is a more rapidly applied stitch for use when multiple tendon divisions require repair.

(3) The interlacing suture (Fig. 84 (d)) is the standard stitch for joining two tendons of unequal diameter.

(4) The Bunnell end-to-end stitch of the withdrawal type (Fig. 85).

(5) The Bunnell stitch for "suture at a distance" (Fig. 86).

(iii) *Insulation of tendon sutures.*—Various materials have been employed to surround the tendon junction in order to prevent adhesion. In the opinion of the writer, free fat, fascial or paratenon grafts, or Aminoplastin, do not serve a useful purpose and it is wiser, if possible, to make use of the tissues which are already available at the site of suture. The paratenon surrounding a free graft

*Prevention of
adhesion
formation*

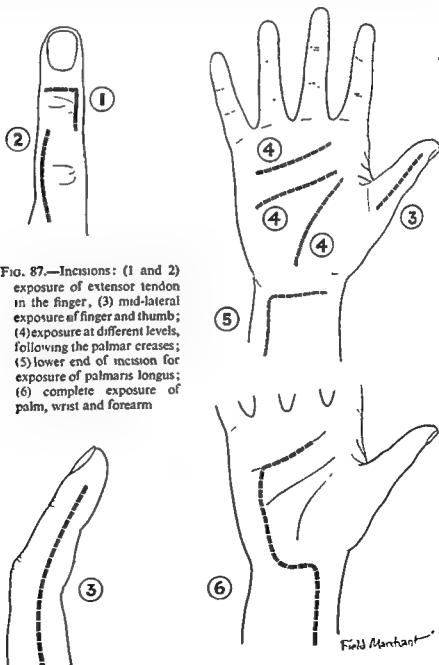


FIG. 87.—Incisions: (1 and 2) exposure of extensor tendon in the finger, (3) mid-lateral exposure of finger and thumb; (4) exposure at different levels, following the palmar creases; (5) lower end of incision for exposure of palmaris longus; (6) complete exposure of palm, wrist and forearm

may be drawn down to cover the suture wire, and when the suture line is in the palm the suture may be buried in the lumbricalis muscle.

(b) Incisions

All incisions should be planned to follow the natural folds of the skin, when this is possible, and should always be designed to avoid crossing a natural flexion crease (Fig. 87). Details of individual incisions are discussed in the special sections.

Following the natural flexion creases

(c) Immediate or delayed repair

It is not only useless but dangerous to undertake immediate tendon repair unless certain fundamental conditions are fulfilled.

(i) The wound must have been caused by a reasonably clean instrument, a wound contaminated by soil or milk is not suitable. Crush injuries are not suitable for immediate repair, since there may be necrosis of tissue beyond the limits detectable by the naked eye.

(ii) Cases in which the injury is complicated by skin loss and by fracture or joint injuries are unsuitable for immediate repair. Nerve injuries are not necessarily a contra-indication, as nerve suture may be performed at the time of operation.

(iii) Treatment must be instituted within a reasonable time of wounding. Six hours is the generally accepted limit, but one must not be bound by a firm rule, and clinical judgement will sometimes extend this arbitrary period.

Importance of early treatment

(iv) Operative conditions must be adequate and the correct instruments and suture materials must be available.

(v) The surgeon must be competent. Flexor tendon repair is difficult work even for the experienced surgeon and, if one is not available, the emergency operation should be limited to a careful wound toilet and skin suture, followed by administration of penicillin. Subsequent elective treatment may be carried out when wound healing is complete.

(4) Injuries to extensor tendons

(a) Distal interphalangeal joint

A mallet-finger deformity may be caused by rupture of the extensor tendon insertion to the distal phalanx, by an avulsion fracture of the base of the phalanx, or by open division of the tendon.

Treatment.—Rupture or division of the tendon may be successfully treated by means of splintage, if such treatment is instituted within the first 3–4 weeks after injury. Splintage must be accurate and should be maintained for 6–7 weeks. Plaster or moulded aluminium may be used and the splint should include the full length of the digit, maintaining the proximal interphalangeal joint in flexion, the distal joint being extended.

Conservative methods

Avulsion fractures are sometimes associated with forward subluxation of the distal phalanx; these cases are well treated by open reduction and fixation of the fragments by stainless-steel wire, the removal-suture technique being used.

Operative treatment

In late cases, tendon rupture, or division, should be repaired by operation. An inverted "L"-shaped or "U"-shaped incision is made and, with small scissors, the tendon is carefully freed from the overlying tissues and from its adhesion to the middle phalanx. It is almost invariably found that union has

occurred, but that there is also lengthening. A small length of 1-2 millimetres of tendon is excised, and a careful end-to-end suture is performed, gauge 40 wire being used. Splintage is maintained for 5-6 weeks, after which movements are instituted, in conjunction with the application of protective splintage designed to prevent undue flexion strain.

Satisfactory results can usually be obtained by this method and, as the only alternatives are an arthrodesis of the interphalangeal joint or a partial amputation, a determined effort to effect repair is justifiable.

(b) Proximal interphalangeal joint

Rupture or division of the central tendon slip attached to the base of the middle phalanx allows the two lateral bands to slip in a volar direction. The proximal interphalangeal joint is thus flexed, and the distal joint is extended, giving rise to the typical deformity.

Conservative treatment

Operative treatment

Treatment.—In early cases of moderate severity, full function can be restored by means of splintage in extension, maintained for 5-6 weeks. In late cases, treatment should be by operation. A curved incision around the side of the joint is used, and one of two repair techniques may be employed. In most cases the central slip can be re-attached by wire to the middle phalanx. In neglected cases this may not be possible, and an alternative method is to release the lateral bands and to suture them together over the dorsal aspect of the joint.

(c) Metacarpo-phalangeal joint and dorsum of hand

Conservative treatment

Tendon division at these levels is not followed by appreciable retraction. A patient who is seen within a week or two of injury may often be treated successfully by means of splintage applied in extension, for 5-6 weeks. In



(a)



(b)

FIG. 88 (a) and (b).—Result obtained after late operative repair of the extensor tendon at the level of the metacarpo-phalangeal joint of the index finger. Skin mark indicates wound

delayed cases, surgical repair is required, with the use of an end-to-end suture and the subsequent application of splintage which holds the metacarpophalangeal joint extended and the interphalangeal joints moderately flexed (Fig 88).

(d) *Injuries to the wrist*

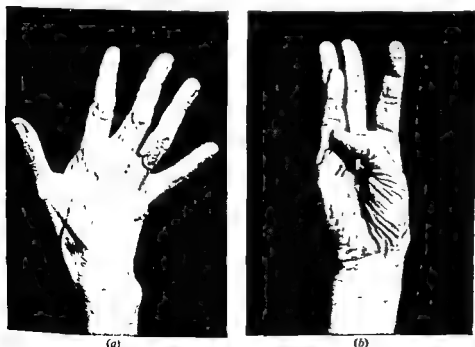
Operative repair is always required in wrist injuries, and the decision to perform an immediate tendon repair in a recent wound, or to perform wound toilet only with a delayed tendon repair undertaken subsequently, must be made for each individual case. *Methods of surgical repair*

In cases in which there is severe destruction and loss of tendon by trauma or infection, the part may be reconstituted by joining divided tendons to intact tendons, or by free graft replacement.

(e) *Attrition rupture of extensor pollicis longus*

Rupture of the extensor pollicis longus at the level of Lister's tubercle is an occasional complication of a Colles's fracture or of a simple posterior cortical crush of the radius. The condition may arise as early as 3 weeks after injury and while the limb is still in plaster, or it may not develop until 3-4 months later. It is easily recognized, and the operation of tendon transference gives most satisfactory results. *Tendon transference*

Direct suture is not recommended, on account of the fraying of the tendon at the site of rupture, and also because further fraying and rupture may occur. The extensor carpi radialis longus and extensor carpi radialis brevis have been used as transfers but the amplitude of movement is insufficient. The extensor indicis proprius is the most suitable tendon to use; it is of the correct size and range of movement, and the index finger rarely suffers by its loss.



(a)

(b)

FIG 89 (a) and (b) —Showing the result obtained after transference of the extensor indicis proprius to the divided extensor pollicis longus. Skin mark indicates wound.

The extensor indicis proprius is divided at its attachment through a $\frac{1}{2}$ -inch transverse incision just proximal to the metacarpo-phalangeal joint, where the tendon lies to the ulnar side of the extensor digitorum communis. The extensor pollicis longus is exposed by a 1-inch transverse incision at the level of the wrist joint. The extensor indicis is located and is withdrawn into the wound. It is joined to the extensor pollicis by an interlacing suture, reasonable, but not exaggerated, tension being obtained. Plaster fixation is employed for 4 weeks and full restoration of function is usually seen in 8-10 weeks (Fig. 89).

(5) Injuries to flexor tendons

Within the digital theca

(i) *Profundus division*.—The profundus tendon may be torn from its insertion by a forcible extension strain of the terminal phalanx, or the tendon may be divided in an open wound. The general function of the hand is not grossly impaired, and in certain cases it is justifiable to accept the disability, particularly if the case is not seen immediately.

If an open division is seen immediately after the injury, the choice of treatment must be guided by the main principles already described. If any doubt exists, it is wiser to perform skin toilet and suture and to deal with the tendon injury when the skin has healed.

Suture

Immediate suture with stainless-steel wire can yield almost perfect results. Delayed suture is also satisfactory, provided that the lapse of time has not been sufficiently long to allow severe retraction and secondary shortening of the muscle to occur. Four weeks is a reasonably safe limit. After this period it is difficult to secure end-to-end apposition, and replacement by a free graft is the more satisfactory procedure.

Replacement by graft

The decision to attempt restoration of the profundus by means of grafting is one that requires careful judgement. It is clearly unwise to perform a standard flexor graft and, in doing so, to remove a good, functioning sublimis, an unsuccessful result carries with it the certainty of a finger rendered useless by interference, and the risk should not be accepted. If a graft is employed, the sublimis must be left undisturbed and a thin graft, for example from the toe extensor, should be used to replace the profundus. Success can be achieved in most cases, but if the result does not come up to expectation, no harm has been done and the finger still has sublimis action.

(ii) *Profundus and sublimis division*.—This injury presents the most difficult tendon problem in the surgery of the hand and it is largely due to the work of Bunnell (1944), Mayer (1938), Koch (1944) and Mason (1940) that the treatment of this injury has been placed on a sound basis.

The beautiful sliding action of the tendons in the digital theca makes one feel doubtful whether restoration of function can ever be achieved by surgical treatment, but it has now been shown repeatedly that excellent results can be obtained provided that accurate and correct methods are employed.

Immediate tendon repair must be undertaken only when ideal conditions are satisfied. When immediate suture is deemed advisable, it is useless to attempt suture of both tendons. Local adhesions binding the tendons together are inevitable with loss of function. The profundus alone is sutured and the sublimis is completely removed. There are two methods of suture available,

Suture

(1) suture at a distance, as described by Bunnell (1944), with subsequent removal of the wire, and (2) local suture with retention of the wire. Bunnell uses 34-35 gauge wire and withdraws it after approximately 3 weeks, when the tendon is physiologically united. For a full description of Bunnell's method, the original work should be consulted.

In those cases in which immediate operation cannot be safely undertaken, on account of the nature of the wound or because of lack of facilities, wound toilet and skin suture alone should be employed; a free tendon graft operation should be performed when the wound is safely healed, all reaction has settled and full passive mobility is restored. Tendon replacement by a graft ensures that the junctions are in safe areas in which adhesions are not likely to occur. Tendon unions between the distal crease of the palm and the proximal crease of a finger will adhere to the digital theca. When tendons are divided in this dangerous area it is wiser to perform a secondary graft operation even though the wound conditions permit primary suture. *Replacement by tendon graft*

(6) Tendon graft operation

(a) Site of graft

Several possible types of graft are available. The palmaris longus is perhaps the one that is most often employed; it is present in approximately 90 per cent of subjects. It is of suitable size and length and can be readily removed. It should be taken with its surrounding paratenon by open dissection and not by the use of a tendon stripper. The sublimis of the same finger may be used, but this tendon is sometimes found to be a little too thick to pass easily through the thecal tunnel over the middle phalanx, and the tendon is not surrounded by paratenon. *Palmaris longus*, *Flexor sublimis*

The long extensor tendons of the second, third, fourth and fifth toes are suitable. These tendons are somewhat thin but they appear to withstand subsequent strain well and, by reason of their small size, they are readily revascularized. They also run easily through the thecal pulleys. *Toe extensors*

The tendons are removed by open dissection with their paratenon, through a curved incision on the antero-lateral aspect of the foot and ankle.

Other tendons that may be used are the plantaris, if it is present, the extensor indicis proprius and the flexor carpi ulnaris, but the last tendon is thick and is not really suitable. *Other tendons*

(b) Technique

The whole length of the digit is exposed by a true mid-lateral incision, which carries the line of dissection posterior to the digital vessels and nerves. The sublimis and profundus tendons are completely removed, except for a small tag of profundus attachment. A pulley is preserved over the proximal and middle phalanges. The palm is exposed by an incision in the proximal palmar or thenar crease and the proximal end of the tendon is drawn into the palm. It is usually found that the proximal end of the tendon is adherent in the blind area between the palm and finger incisions. Considerable effort and patience are necessary to secure its complete removal, which is essential in order to clear the thecal channel. A tendon stripper has been recommended but the writer believes that careful dissection from each end is less traumatic. The channel between finger and palm is then stretched by the use of increasing sizes of Hegar's dilators; the graft is finally threaded through from palm to *Removal of tendon*

finger and then through the pulleys which were previously preserved in the finger.

Sutures

The proximal suture is performed as far proximal in the palm as is practicable. The method of suture depends upon the size of the graft. If the graft and

the profundus tendon are of approximately equal diameters, an end-to-end suture is used. On occasions, when the graft is much smaller than is the profundus, an interlacing suture is used. The distal suture may be made either to the remaining tag of profundus tendon or to the terminal phalanx. It is usually found that the former is the simpler and more satisfactory method.

If it is found to be impossible to use some of the existing fibrous sheath as a pulley, a new pulley may be constructed over the middle phalanx by encircling the bone with a thin strip of tendon

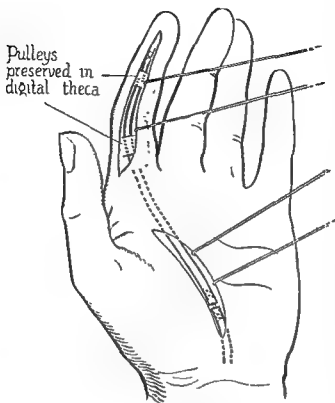


FIG 90—Tendon graft operation for flexor tendon division in digital theca

Tension of graft

The final tension should cause the finger to lie in the correct relationship to the other fingers while under anaesthesia, that is, the index finger is flexed least and the little finger is flexed most. Suture lines should be secure and tested before the final skin suture is performed (Fig. 90).

(c) Post-operative treatment

It is usually recommended that a protective splint holding the wrist in flexion should be employed for approximately 3 weeks and Bunnell (1944) believes that movement should not be permitted for the first 15-21 days. This post-operative treatment is based on the experimental work of Mason and Allen (1941) who claim that active movement, commencing soon after suture, results in a marked tissue reaction, whereas the immobilized tendon shows the least irritative tissue reaction and the least attachment to its surroundings.

The writer has employed several forms of post-operative treatment, varying from immediate gentle active movements to complete immobilization for 2-3 weeks, and is in favour of early movement and a routine in which a wool and crêpe bandage splintage is used, so adjusted as to avoid external extension strain on the finger but permitting flexion to occur. The writer has no doubt

Early movements

that the best results have been obtained by this method. It is, however, with some diffidence that this suggestion is put forward, because of the more generally held opposite view.

Active movements of a gentle nature are permitted from the second day. The hard, blood-stained dressings are changed 5-6 days after operation, and from then onwards gradually increasing movements are permitted. It is exceptional for the suture lines to give way under ordinary active movements,

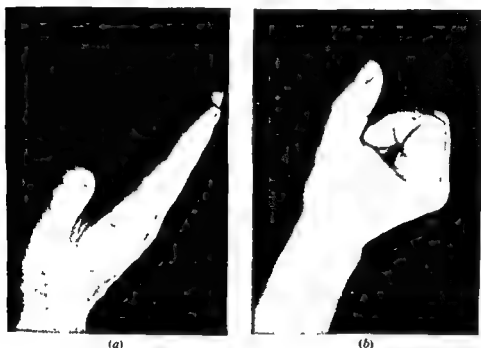


FIG 91 (a) and (b)—Result obtained after a palmaris graft which replaced the superficial and deep flexors of the index finger.

and in only one case has this occurred, but every precaution must be taken to prevent undue passive strains. At the end of 3 weeks more vigorous exercises may be gradually developed and physiotherapy and occupational therapy may be used to encourage movements. The final result cannot be judged until 9-12 months from operation but, in most cases, the patient can return to useful occupation at 4 months (Fig. 91).

(7) Injuries to flexor tendons of thumb and palm

(a) Repair of flexor pollicis longus

The flexor pollicis longus can be sutured directly, with good prospect of success. Immediate or delayed repair should be undertaken according to the conditions of wounding which have been discussed already.

Suture is sometimes impracticable owing either to the passage of time and severe retraction or to marked cicatricial formations. In these cases, either a free graft or one of the two following procedures may be used. The flexor pollicis longus may be lengthened in the forearm by a slide at its musculotendinous junction and 1½-2 inches of extra length, which may permit suture at the terminal phalanx, is obtained. This is a useful technique when division

*Elongation of
flexor pollicis
longus*

has occurred at the level of the proximal phalanx and when retraction prevents direct suturing. In cases in which both muscle and tendon have been destroyed, a complete muscle-tendon replacement is required; this can be obtained by transferring a flexor sublimis unit from a suitable finger to the thumb.

*Transference
of flexor
sublimis*

The exposure of the flexor pollicis longus is complicated by its deep situation in its proximal course. The distal part may readily be exposed by the standard mid-lateral incision; an incision in the thenar crease will reveal the tendon passing between the two heads of the flexor pollicis brevis but more proximally it can be exposed only by division of the transverse carpal ligament. Care must be taken of the median nerve and its motor branch to the thenar muscles.

Incisions

(b) Tendon division in the palm

Tendon division in the palm does not present the difficulties which are encountered within the digital theca, and immediate or delayed direct suture, according to circumstances, can be employed with full expectation of success.

*Sublimis
graft*

*Transference
of flexor
sublimis*

In late cases, in which retraction and lack of muscle elasticity does not permit end-to-end suture, restoration of function may be obtained by bridging the gap in the profundus by means of a free graft taken from the sublimis, or by transferring the flexor sublimis of a neighbouring finger to the distal end of the divided profundus tendon. The donor finger does not suffer appreciably by the loss of its sublimis. It is important not to remove the sublimis near its insertion, since a hyperextension deformity of the proximal interphalangeal joint may occur, causing an objectionable locking of the joint and difficulty in initiating flexion.

Incision

The palm can be readily exposed by an incision in the appropriate palmar or thenar crease. The incision may be extended in a proximal direction from the ulnar side of a palmar crease, if necessary.

(8) Multiple tendon and nerve division at wrist level

Suture

This presents a formidable surgical problem, and opinion differs as to the correct precedence of nerve and tendon suture. Nerve suture is technically easier and more satisfactory if it is delayed for several weeks after division. The nutrition of the hand, however, depends to a large extent on nerve function and will be defective until the nerve is restored. Tendon repair should be performed as soon as practicable, for delay allows adhesions to form; the multiple tendon ends become glued together and muscle retraction renders subsequent end-to-end suture more difficult.

The ideal procedure is to perform a complete nerve-and-tendon suture immediately after the injury, if the wound conditions permit. If the wound conditions are unfavourable or if the surgeon is inexperienced, it is wiser to perform a wound toilet and skin suture only, followed by a formal repair when safe wound healing has occurred.

*Sublimis
grafts*

When repair cannot be undertaken for some weeks or months, it is usually impossible to secure end-to-end tendon apposition, and it is necessary to fill the gaps in the profundus tendon with sublimis grafts.

(9) Tenolysis

The release of a tendon which is bound down by scar tissue resulting from trauma or infection is often followed by remarkable improvement in

function. Before tenolysis is performed, a careful assessment must be made to determine whether there are any complicating factors present. Passive joint mobility must be satisfactory and there should not be any secondary skin contracture. In the case of post-infective adhesions, the risks of awakening a dormant infection must be carefully considered. *Complicating factors*

The operation is tedious but not difficult. It is a common experience to find that the adhesions are more widespread than was expected, and skin incisions must be so planned that they can be enlarged if necessary. The tendon is completely cleared of all adhesions, including the illusive bands which run for some distance alongside a tendon before the attachment to surrounding tissue is discovered.

A proximal exposure of the tendon or muscle should be made in order to determine that a free and unrestricted normal action is present before the operation is completed.

No attempt is made to cover the tendon with transplanted glide material; reliance is placed entirely on the after-treatment for the prevention of further adhesions.

Immediate active movements are essential. The hard, blood-stained dressing is changed a day or two after operation and active movements, assisted by faradism, are commenced. *Post-operative treatment*

The results are usually very satisfactory and at times dramatic in the degree and speed of recovery.

The section on Tendon Injuries in the Hand is based on a Hunterian Lecture (1948) at the Royal College of Surgeons, England.

BIBLIOGRAPHY AND REFERENCES

Primary myositis

Jager, B. V., and Grossman, L. A. (1944) *Arch intern Med*, 73, 271

Gas gangrene

Army Medical Department (1944). *Bulletin (Supplement)*. No 15 London; War Office.

Cantril, S. T., and Buschke, F. (1944) *Radiology*, 43, 333.

Cutler, E. C., and Sandusky, W. R. (1944). *Brit. J. Surg.*, 32, 168.

Godby, W. H. (1940) *Med J Aust.*, 1, 85.

Ham, H. (1940). *Med J. Aust.*, 2, 287.

Jeffery, J. S., and Thomson, M. (1944) *Brit. J. Surg.*, 32, 159.

Macfarlane, M. G. (1945) *Brit. med J.*, 1, 803.

MacLennan, J. D. (1946) *Proc R. Soc. Med.*, 39, 293.

Ogilvie, W. H. (1944) *Brit. J. Surg.*, 31, 313

Smithers, D. W. (1941). *Surgery of Modern Warfare*, p. 122. Edinburgh; Livingstone

Williams, A. J., and Hartzell, H. V. (1939) *West J. Surg.*, 47, 561.

Wilson, J. V. (1946) *Pathology of Traumatic Injuries*, p. 103. Edinburgh, Livingstone

Trichinosis

Manson-Bahr, P. H. (1939) In *British Encyclopaedia of Medical Practice*, Vol. 12, p. 241. London; Butterworth

Suessenguth, Hazel, and Kline, B. S. (1944) *Amer J. clin. Path.*, 14, 471.

- Jones, R., and Lovett, R. W. (1929). *Orthopaedic Surgery*, 2nd ed., ■ 550. London; Milford
- McMurray, T. P. (1937). *Practice of Orthopaedic Surgery*, p. 350. London; Arnold
- Watson-Jones, R. (1943) *Fractures and Joint Injuries*, 3rd ed., p. 772. Edinburgh; Livingstone.

Tendon injuries in the hand

- Bunnell, S. (1944) *Surgery of the Hand*, pp. 277 and 449. Philadelphia; Lippincott.
- Graham, W. C. (1947). *J. Bone Jt Surg.*, 29, 553.
- Kinmonth, J. H (1947) *Brit J. Surg.*, 35, 29.
- Koch, S. L. (1944). *Surg. Gynec. Obstet.*, 78, 9
- Luckey, C. A., and McPherson, S. R. (1947). *J. Bone Jt Surg.*, 29, 560.
- Mason, M. L. (1940). *Surg. Gynec. Obstet.*, 70, 392
- and Allen, H. S (1941). *Ann Surg.*, 113, 424.
- Mayer, L. (1938). *Amer. J. Surg.*, 42, 714

[References to other titles are given under Muscle and Tendon—Diseases and Injuries, in the Index Volume. The subject is also dealt with under the heading of Muscle Diseases in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 11.]

NECK-CELLULITIS

BY IVOR LEWIS, M.D., M.S., F.R.C.S.

SURGEON AND MEDICAL DIRECTOR, NORTH MIDDLESEX HOSPITAL, LONDON

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1. DEFINITION AND AETIOLOGY

236.] Infection of the subcutaneous and connective tissue spaces of the neck may occur at all ages, but is commonest in children owing to their proneness to infection of the scalp, of the throat and of the ears. Young adults are also frequent sufferers because of sepsis of the teeth and jaws. With increasing age man loses his teeth, and his pharyngeal lymphoid tissue retrogresses; thus two of the main causes of cellulitis of the neck disappear.

2. SURGICAL ANATOMY AND PATHOLOGY

Cellulitis of the neck may be divided into infections superficial to the deep cervical fascia and those deep to it. The deep fascia in the neck is well defined and of importance in limiting the spread of infections; still oftener its importance is due to its guiding and leading the infection to distant and dangerous areas while effectively preventing it from reaching the surface.

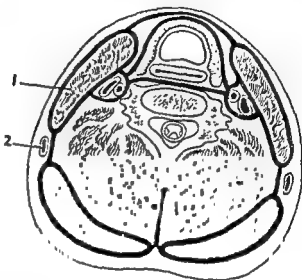
The *investing layer* encloses all the neck structures except the superficial vessels and nerves and the platysma muscle. Superiorly it is attached to the inferior margin of the mandible, behind which it splits to enclose the parotid gland and is attached to the zygoma, the mastoid process and the superior nuchal line. In the middle line posteriorly it is attached to the cervical spinous processes, and encloses the trapezius muscle. Laterally it splits to enclose the sternomastoid muscles. Below it is attached to the sternum and the clavicles.

The *pre-vertebral fascia* is a dense septum dividing the neck into its osseomuscular and visceral compartments. It forms the floor of the supraclavicular triangle. The brachial plexus pierces the fascia taking a tubular investment

*The cervical
fascial planes*

with it into the axilla. It lies in the posterior triangle of the neck. It has close connexions with the facial and parotid fascial spaces, as well as with the mastoid process by way of the internal jugular vein (Fig. 92). Whereas in some cases it

FIG 92.—Section of neck showing disposition of the deep fascia 1 = Sternomastoid muscle. 2 = External jugular vein.



acts as a protective sheath of the neck, in other

The following are :

(1) The investing layer, by splitting above to enclose the bellies of the digastric muscles, and by adhesion to the hyoid bone, tends to prevent infections in the submandibular triangles from spreading down the neck. Thus in most cases infections of the floor of the mouth and of the submandibular and retromandibular spaces keep to the rather intricate anatomical planes of this region. Four spaces may usefully be considered—pus in any of them is apt to communicate with the others, and all of them may drain into the carotid sheath. Two of these spaces are placed anteriorly, these are the submandibular space and the sublingual space (deep). Two are placed posteriorly and are the parotid space and the pterygomandibular space (deep).

Spaces in floor of mouth and upper part of neck

The submandibular space is most often infected from the lymphatic glands or from the submandibular salivary gland. The floor of the space is formed by the mylohyoid muscle and the roof by the investing layer of the deep cervical fascia as it splits to contain the submandibular salivary gland. It communicates around the posterior border of the mylohyoid muscle with the sublingual fascial space. Posteriorly it is limited by a process of deep cervical fascia.

The sublingual space is the areolar space between the tongue and the muscular floor of the mouth—the mylohyoid muscle. It is bounded above by the hyoglossus muscle and the tongue, by the mylohyoid muscle below,

and by the jaw laterally. It contains the sublingual gland, the submandibular duct and deep process of the gland, and the geniohyoid muscles.

The *parotid fascial space* is formed of deep cervical fascia. Infection arising in the gland may, however, track into the carotid sheath, or rupture into the external auditory meatus to which the fascia is attached (Fig. 93).

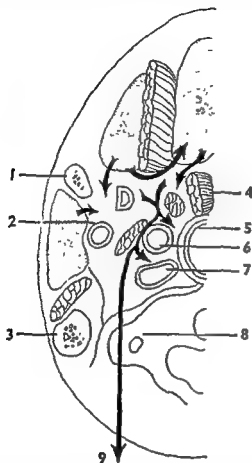


FIG 93—Schema of four compartments in the floor of the mouth and the upper neck, showing routes along which pus may track. A = Submandibular space, separated by mylohyoid muscle from B = sublingual space; C = Parotid fascial space; D = Pterygomandibular space. 1 = Mandible. 2 = Posterior facial vein. 3 = Mastoid process. 4 = Internal pterygoid muscle. 5 = Pharynx. 6 = Internal carotid artery. 7 = Internal jugular vein. 8 = Transverse process of first cervical vertebra. 9 = Route by which pus often tracks down the neck along the carotid sheath.

The *pterygomandibular space* is bounded anteriorly by the posterior border of the mandible and by the pterygoid process, posteriorly by the mastoid process and the transverse processes of the atlas and axis. It is roofed by the petrous portion of the temporal bone and by the external auditory meatus. The superior constrictor and the internal pterygoid muscles line the space anteriorly, and, below, it is separated from the carotid triangle by the posterior belly of the digastric muscle. Infections of the molar teeth and of the tonsils and pharyngeal lymphatic glands easily gain access to this space, whence they may spread downwards along the carotid sheath. Moreover, pus in the submandibular and sublingual spaces may extend into the pterygomandibular space from the front.

(2) Infections behind the pre-vertebral layer must spread either laterally or distally. Pus from caries or osteomyelitis of the cervical spine thus tracks laterally along the brachial plexus, or else distally into the mediastinum in front of the pre-vertebral muscles.

(3) The great majority of other deep infections of the neck, whether arising from lymphatic glands, disease or perforation of the viscera, from the great

vessels, or the thyroid gland, will collect in the *visceral space*. This space is capable of much distension, otherwise spread into the mediastinum would be almost inevitable. *Visceral space*

(4) The carotid sheath is one of the most lethal conduits of infection in the body, liable to carry virulent infections from tonsils, mastoid and jaw down the neck into the mediastinum.

The commonest organisms in the acute infections are the *Staphylococcus aureus* (if arising from skin infection or osteomyelitis), and the streptococcus (especially from infection of the fauces). A mixture of the above, with anaerobes, may be found from perforation of the oesophagus and from infected wisdom teeth and always results in most dangerous infections. *Bacteriology*

Subcutaneous cellulitis differs little in the neck from a similar condition in other parts. Owing to the frequency of skin infections in the scalp, face and neck itself (such as impetigo, acne and scratches) and of carbuncle of the nape, subcutaneous cellulitis of the neck is often seen. The subcutaneous tissue on the back of the neck is thick and fibrous, and sloughs are often formed.

3. CLINICAL PICTURE

As elsewhere, the severity of the illness will vary with the virulence of the infection and with the tension which it is under. But in deep infections of the neck the ease and rapidity of spread often spells a fulminating course. Patients with severe infections of the neck may be gravely ill with high intermittent fever, rigors and delirium. Often, however, the symptoms are less violent, the temperature may not be high, and there may be only moderate swelling and slight pain. An increasing tachycardia and a failing circulation may be followed by pain in the chest, dyspnoea, cyanosis and terminal broncho-pneumonia. In the majority of cases the infection is less severe and becomes limited; local swelling develops, together with the signs of an abscess. The other features depend upon the situation and origin of the trouble. Acute cellulitis of the submaxillary space usually becomes well localized but it may spread to the space deep to the mylohyoid muscle (Ludwig's angina) or cause oedema of the glottis. Infections of the lymphatic glands are the least liable to spread to the mediastinum, and those from perforation of the oesophagus the most liable. In the latter case there will be a history of an impacted foreign body or of instrumentation. Even a Ryle's tube left in continuously for days has caused perforation necrosis of the oesophagus. Infection may occasionally arise in the thyroid gland or in the laryngeal cartilages, and this may give rise to a most difficult clinical picture. Bone infection, whether of mandible, mastoid or cervical spine, will generally present signs, clinical and radiological, except in acute osteomyelitis when the skiagrams are normal for the first week. The inflammatory swelling may cause difficulty in swallowing or in breathing, according to its site. Although I have once seen an acute abscess of the neck of a child cause a Horner's syndrome, nerve palsies are exceptional. Surgical emphysema is occasionally seen and, whether due to visceral perforation or to gas-producing organisms, is of grave significance.

Ludwig's angina

Abscess of the sublingual space between the hyoglossus and mylohyoid is a serious condition and is associated with difficulty in opening the mouth and

with great pain. There is often oedema of the pharynx and of the aryepiglottic folds. Externally there is a bilateral brawny swelling of the floor of the mouth. Submandibular abscess, on the other hand, is almost always unilateral and generally tends to become localized, although it also may extend back into the pterygomandibular space, or around the posterior border of the mylohyoid muscle into the sublingual space—chiefly when the infection arises in the submandibular salivary gland.

Deep abscesses may bulge into the pharynx—especially those of the pterygomandibular space which project through the postero-lateral wall of the pharynx. The better-known acute retropharyngeal abscess bulges posteriorly and is due to suppuration in the retropharyngeal lymphatic glands. More rarely a retropharyngeal abscess behind the pre-vertebral fascia, due to acute osteomyelitis of the cervical spine or base of the skull, may do likewise; it is important to remember this in the differential diagnosis. A puzzling lesion of the thyroid region is that sometimes called Reclus's disease or woody phlegmon. It is almost impossible clinically to differentiate it from advanced diffuse carcinoma of the thyroid, indeed some of the cases turn out to be malignant with supervening inflammation.

Now and again an acute cellulitis of the neck—usually of glandular origin—will not be seen by the surgeon until it has formed a chronic abscess. It may then be indistinguishable from a tuberculous abscess until the pus is examined. Similarly, attacks of inflammation often make a branchial cyst resemble a chronic or subacute abscess.

4. SPECIAL AIDS TO DIAGNOSIS

(1) Radiography

Good lateral films of the neck may show foreign bodies, soft-tissue swellings deforming the outline of pharynx or trachea, surgical emphysema, bone disease in teeth, jaws or spine, or calcified tuberculous glands. Skiagrams of the chest may show complicating mediastinitis with widening and filling in of the cardiophrenic angle.

(2) Endoscopy

Endoscopy may show swelling, a foreign body, trauma or underlying new growth.

(3) Pathological investigation

A blood culture should be made, especially if rigors are occurring, and a blood count may give some indication of localization. Aspiration should be performed and pus examined for its cytology and bacteriology.

5. DIFFERENTIAL DIAGNOSIS

(3) bones and cartilage—spine, mastoid, mandible, septic teeth; (4) vascular perforation by foreign body, thyroiditis; (5) phlebitis; (6) infected wounds; (7) infected neoplasms—carcinoma and branchial and thyroid cysts.

The site of the lesion will, in the first place, indicate which origin is most likely. The history will indicate the diagnosis in cases of instrumentation or

Retro-pharyngeal abscess

Reclus's disease

operation. A perforation of the oesophagus, from instrumentation or from impaction of swallowed bones, is about the level of the sixth cervical vertebra (crico-pharyngeus). It can be confirmed by oesophagoscopy—pus will usually be seen exuding from the perforation. When it is difficult to tell a tuberculous infection from a pyogenic cellulitis, a skiagram showing shadows of calcified glands will often indicate the diagnosis. Aspiration of the fluid and the bacteriology and cytology of the pus should decide the question. Careful examination of Wharton's ducts will always show a turbid efflux in sub-mandibular salivary gland infection. Scrutiny of the teeth and of the alveolar process, by inspection, palpation and skiagrams, will reveal infections from this cause. Septic phlebitis—usually of the internal jugular vein—may be found in mastoid and faucial infections. There is tenderness on deep pressure over the vein and often there are rigors. Fluid aspirated from a branchial cyst will contain cholesterol crystals.

In practice some breaking-down growths such as carcinoma of the thyroid gland, or secondary cancer of glands, show all the signs of inflammation. The history alone is likely to prevent an unnecessary exploration. Inflamed cysts, branchial or thyroglossal, or thyroid cystadenoma, may baffle the most experienced clinician. An interesting condition, which at first sight has often been called cellulitis of the neck, is superior vena caval obstruction due to a mediastinal tumour. This causes a tense bull-neck which, however, is neither hot nor tender. There are large veins over the chest, and percussion, or better still radiography, will reveal the lesion. *Diffusent neoplasm*

6. PROGNOSIS

The prognosis naturally varies with the cause. Superficial cellulitis is not appreciably more dangerous than a similar lesion in other parts of the body. The seriousness of deep cellulitis varies not only with the cause, but also with the fascial compartment in which it is found. By far the most dangerous is the space in front of the pre-vertebral fascia, leading down with no resistance, except for the flimsiest areolar tissue, into the mediastinum. The carotid sheath is a channel of almost equal peril. As for the source of the infection, the most dangerous is perforation of pharynx = oesophagus. Certain fulminating infections from the tonsils or from impacted wisdom teeth come a close second.

The prognosis has been transformed by penicillin. Before its introduction perforations of the oesophagus were almost invariably fatal. Septic phlebitis in the neck from it.

with penicillin and severe cellulitis will then be seen less often.

7. TREATMENT

The treatment of cellulitis of the neck has been revolutionized by the introduction of penicillin. The sulphonamides also were of value in some of the haemolytic streptococcal infections, but now are hardly used at all if penicillin is available.

The essential treatment of deep cellulitis of the neck, from whatever cause, is penicillin, operative treatment taking a second place. Penicillin should be

given by 3-hourly intramuscular injections of at least 30,000 units—more in fulminating infections. It is unwise to trust to less frequent but massive doses—they are wasteful and usually less efficient. Penicillin should be continued for some days after the fever has subsided.

8. OPERATIONS

Before any operation the patient should be on full penicillin treatment. Operative treatment may be classified as follows.

(1) Removal of the cause

It is generally sound surgery to remove any gross cause, for example, the impacted bone which may still be in the oesophagus, or partly in and partly through its wall. Removal will prevent further damage and particularly will allow peri-oesophageal pus to drain back into the oesophagus, often with surprising adequacy. For other foreign bodies the same advice applies.

By way of prophylactic surgery it is a good plan, if a perforation of the oesophagus is recognized at the time, to cut down on it, and either suture it carefully or place a drain down to it, leaving the wound wide open.

(2) Drainage of abscesses

Abscesses will form fairly commonly and will need incision. It cannot be repeated too often that fluctuation is not to be expected in these abscesses of the neck, but rather a brawny localized swelling. With penicillin, they are best left until they are really well localized, bearing in mind, however, that an

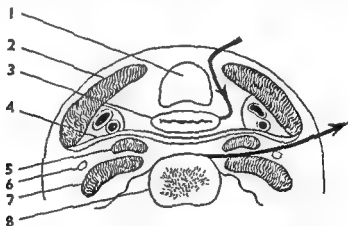


FIG. 94.—Transverse section of neck at level of sixth cervical vertebra with schema of operative routes to two main sites of pus deep in the neck. 1 = Trachea. 2 = Sternomastoid muscle. 3 = Oesophagus. 4 = Pre-vertebral fascia. 5 = Scalenus anterior muscle. 6 = A cervical nerve. 7 = Scalenus medius muscle. 8 = Body of cervical vertebra.

abscess left too long may burst into the pharynx or mediastinum. Under this régime the technique required will be of the simplest, few formal procedures being needed, and the only precaution will be to avoid important vessels and nerves in making the incisions (Fig. 94). It is a good practice to incise only the skin and deep fascia, using a sinus forceps for the deeper layers. Corrugated-rubber drains rather than tubes should be used for drainage.

(a) *Special sites*

(i) *Lateral pharyngeal abscess*.—An acute lateral pharyngeal abscess of faucial origin is best opened into the pharynx under local anaesthesia with a sinus forceps in just the same way as a peritonsillar abscess.

(ii) *Acute retropharyngeal abscess of faucial origin*.—This abscess should be opened in the same way as a lateral pharyngeal abscess.

(iii) *Acute abscess from osteomyelitis of spine*.—This should be drained externally through the posterior triangle, a track being made with a blunt forceps between the scalenus medius and scalenus anterior muscles, behind the carotid sheath.

(iv) *Chronic retropharyngeal abscess due to tuberculosis of the spine*.—This should not be drained but, if bulging, it may be aspirated through the posterior triangle, and the neck immobilized in plaster.

(v) *Ludwig's angina*.—In the treatment of Ludwig's angina there is no longer any place for the heroic surgery once advocated, such as wide and early incision with total removal of the submandibular salivary gland. Penicillin is the sole treatment required in most cases. The pus will often rupture into the floor of the mouth. If a localized swelling persists, it is best opened by a moderate incision—an inch or more long, either transversely 2 inches behind the chin or, better, halfway between the midline and the inferior border of the mandible. This must go through the deep fascia and the mylohyoid muscle and must open its fibres widely. A corrugated drain is stitched in.

(b) *Anaesthesia*

Local infiltration anaesthesia is the safest, but unless the abscess is pointing, an intravenous anaesthetic is kinder, with passage of an endotracheal tube beforehand. It must be emphasized again that an inhalation anaesthetic is to be given for operation on any of these acute infections of the floor of the mouth or upper neck only if an anaesthetist who is expert at tracheal intubation is available. Even so, intubation under local anaesthesia followed by an intravenous anaesthetic is safer, and the safest of all is a local anaesthetic.

Whatever the anaesthetic used, *tracheotomy instruments must always be on the table*.

(3) *Operations for complications*

Tracheotomy should seldom be required except to relieve unexpected suffocation during an anaesthetic. Whenever there is reason to suspect oedema of the glottis the surgeon will employ a local anaesthetic, or failing that, a general anaesthetic with an endotracheal tube *in situ*. This should be left in the trachea until the patient is conscious. Gastrostomy is occasionally helpful in bad lesions of the oesophagus, if swallowing is likely to be impossible for a week or more. In many such cases, however, feeding by an oesophageal tube will meet the case.

9. POST-OPERATIVE CARE

Systemic penicillin should be continued for some days—even a week or two—after the operation. Dressings should be as infrequent as possible, but will need to be done once a day if the patient is to be comfortable. Drains should not be left in longer than three days lest they cause a secondary haemorrhage.

An exception to this is a drain leading down to a perforated hollow viscus, in which case it is best left in for at least a week. It does not matter what local application is applied provided it is sterile.

It is usually best to avoid hot fomentations after operation, though on occasion they may relieve pain and may comfort the patient greatly. Some surgeons have advocated the enclosure, after operation, of the whole neck in a plaster-of-Paris case; this has two serious drawbacks—it is uncomfortable and messy, and it makes it impossible to inspect the area. During the post-operative period a close look-out must be kept for pulmonary complications—bronchopneumonia and abscess of the lung. It is probably unwise to carry out respiratory exercises until the patient has recovered. The heart must be examined, and the spleen felt for, during convalescence, and the slightest return of pyrexia must be tracked down, and if necessary a blood culture carried out.

The scars left by multiple incisions in the neck are apt to be unsightly, but after a year or so they become much less obvious, especially if the incisions have been directed along the tension lines of Langer. Occasionally it may be justifiable to excise some scars in due course in order to secure a better cosmetic effect.

10. RESULTS OF TREATMENT

It is unprofitable to attempt to state any figures for infections of such diverse origin and nature. It may be said, however, that even in the gravest cases, the results at the present time, thanks to penicillin, are remarkably good. The mortality rate of yesterday is the recovery rate of today, provided penicillin treatment is commenced early and is adequate, and provided operation is deferred until the pus is really localized.

[References to other titles are given under Neck—Cellulitis, in the Index Volume]

NECK—CUT THROAT

BY IVOR LEWIS, M.D., M.S., F.R.C.S.

SURGEON AND MEDICAL DIRECTOR, NORTH MIDDLESEX HOSPITAL, LONDON

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1. DEFINITION AND AETIOLOGY

237.] In times of peace, wounds of the neck are seldom accidental—no doubt due, in large part, to the instinctive protective flexion that occurs in an emergency. The wounds are usually suicidal, but are occasionally murderous. Not long ago this was the commonest method of suicide for man. It is now, at any rate in Great Britain, less common than coal-gas and other forms of poisoning, and shooting. There is probably no other kind of attempted suicide which can be so effectively treated as cut throat, provided that the patient reaches hospital alive and that a few simple rules are intelligently applied.

2. SURGICAL ANATOMY

The simplest way of considering these wounds of the front of the neck is according to their depth, direction and level. The determination of the suicide seems to vary in different countries; of British patients who reach hospital alive, in only about half the number has the wound penetrated the air passages. The carotid arteries and the internal jugular vein, which are the suicide's objective, are deeply placed, particularly on extension of the neck for the fatal stroke, because they receive cover from the laryngeal cartilages and the sternomastoid muscle. It is of medico-legal importance that the surgeon should make careful notes of the external appearances and measurements in any wound of the throat. Details should be obtained from books on medical jurisprudence, but it is worth repeating that self-inflicted throat wounds (1) are sometimes multiple and superficial, (2) slope downwards from left to right in a right-handed man, and are deeper on the side on which the incision starts, and (3) often have subsidiary tentative incisions near the main wound.

The level is almost always in the upper part of the neck—three-quarters of the wounds are over the larynx, either above or below the vocal cords. A small minority involve the upper end of the trachea. The pharynx is opened in wounds above the thyroid cartilage. The oesophagus is hardly ever opened. It is encouraging that the trachea or the larynx is seldom completely divided. The epiglottis is frequently cut in thyrohyoid wounds. The structures commonly wounded are: (1) *viscera*: pharynx (often), larynx (often), thyroid gland (often), trachea (occasionally); (2) *vessels*: the external jugular veins,

and the superior thyroid vessels are the common vessels to be involved—sometimes the facial vessels in suprahyoid wounds; very rarely a patient with a wounded carotid artery or internal jugular vein may reach hospital alive;

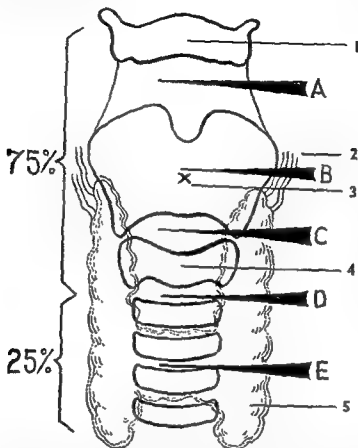
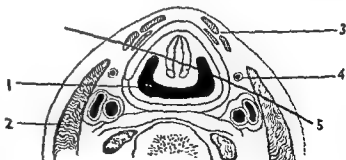


FIG. 95.—Five common sites of wounds in cut throat (A, B, C, D and E). 1 = Hyoid bone 2 = Superior thyroid vessels. 3 = Level of vocal cords. 4 = Cricoid cartilage 5 = Thyroid gland.

(3) *nerves*: usually none of importance is involved. The laryngeal nerves, both superior and recurrent, escape; (4) *muscles*: infrahyoids, cricothyroid, sternomastoid (Figs. 95 and 96). The wounds are generally clean; the chief

FIG. 96.—Diagram of cut throat at infrahyoid level. 1 = Pharynx 2 = Sternomastoid muscle 3 = Infrahyoid muscles. 4 = Superior thyroid artery. 5 = Line of typical wound.



contamination is from the open pharynx or larynx, for streptococci and other virulent organisms are often present in the escaping mucus.

3. CLINICAL PICTURE

A certain number (say, 20 per cent) of these wounds are so grave that the surgeon does not see the patient alive. Usually the patients will be found to

be cold, pale and quiet, their clothes drenched with blood. Though conscious they are unable or unwilling to give much account of the incident—unless the attack happens to have been a murderous one. The most serious aspect is the loss of blood. This, however, has generally ceased by the time the patient reaches hospital, exsanguinated or even pulseless. About half the number of wounds penetrate the air or food passages. There is often obstructed or noisy breathing through the wound, or bubbly tracheal noises from blood flowing into the trachea. Also, when the pharynx is opened, there is danger of the aspiration of blood and even of suffocation. Air embolism is the cause of death in some cases. Cases of cut throat seldom arrive for admission to hospital later than the first few hours, but if admitted late they may already have signs and symptoms of aspiration pneumonia.

4. DIAGNOSIS

Only the gentlest examination should be made before the patient is on the operation table, as anything more is likely to start the bleeding again. One should, however, aim at: (1) assessing the severity of the loss of blood and the fitness of the patient for operation; (2) obtaining a rough idea of the level of the wound, and whether the food or air passages or both are opened; (3) noting any signs pointing to suicide or murder; (4) noting any cause of respiratory embarrassment; and (5) discovering whether any bleeding is still going on.

5. PROGNOSIS

Prognosis varies greatly, but in Great Britain probably about 20 per cent of these patients do not reach hospital. Of those who do, about 30 per cent are likely to die from haemorrhage, broncho-pneumonia and lack of the will to live.

6. PRE-OPERATIVE CARE

Much argument has been heard as to whether or not it is justifiable to use large quantities of blood to revive some poor wretch who has found life more than he can bear. It is best for the surgeon not to try to judge in these matters, but to use blood, if reasonably available, in just the same way that he would use other resuscitative procedures. Apart from transfusion the patient is given Omnopon and scopolamine as a pre-operative sedative; any obvious bleeding point is picked up with forceps; the wound is not disturbed at all unless respiration is embarrassed by the wound itself or by bleeding into the trachea. In some of these cases it is easy and advantageous to pass a tracheal tube through the wound, and leave it in until the operation begins; a fairly firm dressing can then be applied around the neck.

7. OPERATIVE TECHNIQUE

As most of the patients have in fact already ceased bleeding, it is better not to disturb them until they are on the operation table. In a few, local anaesthesia is all one needs—but usually a general anaesthetic is preferable. A small amount of intravenous hexobarbitone for the induction, followed by intra-

Viscera

without any coughing or struggling is essential if further bleeding is to be avoided. The wound should be enlarged, if necessary, to get a good view of the injured structures.

The pharyngeal wall should be carefully and accurately apposed, and sutured with fine interrupted sutures. Fine silk is best, provided that the needles are fine and non-cutting, the sutures interrupted and their knots placed inside the lumen of the pharynx. The epiglottis is often wounded—if badly it may be cut off, if slightly it may be sutured. The larynx and trachea must be sutured with even greater care, and it is probably best to use submucous interrupted sutures of fine catgut. Most surgeons condemn sutures through the cartilages themselves as they are apt to cause necrosis. Sometimes, however, as when the cricoid cartilage is divided across, there is no alternative but to suture the cartilage, or at any rate the perichondrium, if the larynx is to be prevented from collapsing.

Tracheotomy

If there is anything more than the slightest opening in the trachea or in the larynx below the cords, then a tracheotomy should invariably be done. Also invariably, the tracheotomy should be done, *not through the wound*, but as a new and deliberate low tracheotomy. After dividing or pulling upwards the thyroid isthmus, a disc should be removed from the anterior tracheal wall just large enough to take the tracheotomy tube. Wounds of the larynx will lead to stricture in practically every case if a tube is left in the laryngeal wound itself. Moreover, if a low tracheotomy is done as the first step in these cases, the anaesthetic can be continued through it, the larynx and pharynx packed off, and the operation completed with comfort and precision.

Vessels

The external jugular vein is the common source of severe haemorrhage. All wounded vessels should be sought and carefully tied—the vessel and nothing but the vessel. Even though it has ceased bleeding, it should nevertheless be tied. This lessens the chances of recurrent haemorrhage from coughing, and probably also reduces the risk of secondary haemorrhage. The only vessel which should not be securely tied forthwith is the *internal* carotid artery. If the wound of this artery is small and bleeding can be controlled by suture or pressure, it is best to temporize. The common or external carotid arteries may be safely tied without causing hemiplegia. The internal jugular vein can also be tied.

Wounds generally

No formal excision is indicated for these wounds, as they have been made with a sharp instrument and the soiling is only slight. Untidy tags and shreds should be cut off—that is all. Small loose fragments of cartilage are best removed. Muscles and fasciae are sutured with interrupted fine catgut. A corrugated drain should be left down to the mucosa if the pharynx or larynx has been opened, regardless of the separate tracheotomy. In clean and early cases the wound may be closed around a drain, but not too tightly. In septic cases, or any cases not operated on until after 12 hours, a special point should still be made of meticulously closing the larynx and pharynx, preferably with fine silk. The rest of the wound can then be sutured very loosely or, better, packed lightly with Vaseline gauze. A tracheotomy is performed in these cases also. Finally, if the condition of the patient and the experience of the surgeon warrant it, a bronchoscopy should be done before the patient leaves the table. In this way the bronchi can be thoroughly cleansed, and, incidentally, a view obtained of the apposition of the laryngeal wound from the inside.

*Infected cases**Bronchoscopy*

8. POST-OPERATIVE CARE

Further transfusions will often be needed. The patient must be encouraged to cough regularly. Systemic penicillin may be indicated for some days. Sometimes further bronchoscopy will be necessary when there is massive collapse from the aspiration of blood. Broncho-pneumonia and lung abscess are common complications—penicillin has a most important place in their treatment. Secondary haemorrhage is also rather common—it sometimes kills the patient by suffocation if the internal wound has not healed.

Drains should be out by the third day, sutures by the tenth day. The most important point, however, is when to remove the tracheotomy tube. If the laryngeal injury is slight and well away from the vocal cords, three weeks is probably long enough, whereas in the severer injuries, especially in those near the cords, even three months may be too soon. In all cases, careful trial is necessary before decision is made. Much guidance may be given by laryngoscopy in judging the healing internally.

9. RESULTS OF TREATMENT

Whatever the outcome, these cases are apt to be disappointing. If the patient dies, it is disappointing to the surgeon—if he recovers, disappointing to the patient. Moreover, in many of the severer laryngeal wounds, stricture of the larynx will develop—a trial and a disappointment to both. Finally, it is accepted by alienists that a majority of those who have made a determined but unsuccessful attempt on their lives will sooner or later make a successful one.

Blood transfusions and penicillin have reduced the mortality and morbidity. Sinuses are not uncommon—from sutures, from necrosing cartilage or from a fistulous opening internally.

Stricture of the larynx is the most serious late complication, and the question of decannulation is intimately bound up with it. Stricture often develops quite late—from three to six months after the wounding. It is held by many that the only safe way to manage these wounds of the larynx is to leave a (rubber) tracheotomy tube in for six months or more. In the less severe case in which the tube has been removed early, the patient should attend for laryngoscopy every two or three weeks for at least a year.

[References to other titles are given under Neck—Cut Throat, in the Index Volume.]

NECK—CYSTIC SWELLINGS OF

BY FRANCIS A. D'ABREU, CH.M., F.R.C.S.
ASSISTANT SURGEON, WESTMINSTER HOSPITAL, LONDON

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1. GENERAL CLASSIFICATION

238.] Cysts of the neck are classified into developmental and acquired types. The developmental cysts are traditionally divided into midline and lateral. The acquired cysts, such as sebaceous cysts, implantation dermoids and parasitic cysts, may occur in the neck as elsewhere in the body.

2. MIDLINE DEVELOPMENTAL CYSTS

(1) Sublingual dermoid

(a) Aetiology

Towards the end of the third week of embryonic life, five branchial arches are formed between the mouth and the pericardium. These arches are

separated by pouches—the branchial clefts. The arches grow ventrally towards the midline, and imperfect closure of the first and second gives rise to the sublingual dermoid cyst.

(b) *Surgical anatomy*

The cyst lies upon the mylohyoid muscles and separates the two geniohyoids whose medial edges normally join in the middle line. In a similar way the cyst, if large, may also separate the two genioglossi. The upper surface of the cyst lies under the mucous membrane of the anterior part of the floor of the mouth.

(c) *Pathology*

The cyst lining is usually epidermal, but may occasionally be of mucous membrane. If the first, its contents are the usual cheesy, sebaceous material of a dermoid; if the second, the contents are fluid.

(d) *Clinical picture*

The sublingual dermoid, in common with other developmental cysts of the neck, usually begins to grow in early adult life and is not noticed until then. It forms a fluctuant yellowish swelling in the midline of the floor of the mouth. The surface is smooth and dome-shaped, and if the cyst reaches a large size it projects not only into the mouth but into the submental region. It can then be easily palpated between a finger placed underneath the tongue and another pressing upwards under the chin.

(e) *Differential diagnosis*

A ranula is a bluish, thin-walled mucous cyst in a more lateral situation in the floor of the mouth. Its outline and surface are more regular and the swelling is much softer. Submental adenitis and abscess formation in infected submental glands might simulate a sublingual dermoid, but it will be tender and show the usual signs of inflammation. *Ranula*
Submental abscess

(f) *Treatment*

Sublingual dermoids will, if left, grow larger and become more and more uncomfortable. They should be removed in all cases.

(g) *Operative technique*

With the shoulders supported on a pillow in order to extend the neck, an incision is made transversely beneath the chin over the swelling, dividing skin, fascia and platysma. The median raphe dividing the mylohyoids is then split vertically and the cyst dissected out. This dissection is easy, but if the cyst is very large an opening can be made and the contents evacuated in order to facilitate removal. If the floor of the mouth is opened during the dissection, it should be closed with catgut stitches. The mylohyoids and skin are closed around a small drainage tube.

(2) Thyroglossal cyst

(a) *Aetiology*

These cysts occur at any point along the thyroglossal tract, which extends from the foramen caecum at the back of the tongue to the isthmus of the thyroid gland. The thyroid develops from the floor of the pharynx from a depression just below the tuberculum impar, which lies in the midline between the first and second arches. This depression extends caudally as a tubular duct

which bifurcates and forms the thyroid gland. Imperfect closure of this duct is said to give rise to a thyroglossal fistula or a thyroglossal cyst, and may occur at any point along its extent. Cysts below the isthmus of the thyroid are also described as being of thyroglossal origin, but probably originate in the thymic tract.



FIG. 97.—Diagram showing sites of thyroglossal cysts.

(b) *Surgical anatomy*

The cysts are divided into: (1) subhyoid, (2) suprahyoid, and (3) lingual cysts (Fig. 97).

(1) The subhyoid position is the most common site, between the isthmus of the thyroid gland and the hyoid bone. Here they lie beneath the suprahyoid muscles and upon the thyroid cartilage or thyrohyoid membrane.

(2) Suprahyoid cysts are less common; they lie deep to the mylohyoid muscles and below the base of the tongue.

(3) Lingual cysts are extremely rare; they are situated in the substance of the tongue and just beneath the foramen caecum.

(c) *Pathology*

The cyst lining is usually a mixture of columnar, sometimes ciliated, and squamous epithelium. The contents are mucoid and are usually clear but in some cases may be turbid from admixture with cholesterol and altered blood. They may become infected, and discharge through the skin to form a thyroglossal fistula. Such a change is, however, more often due to imperfect removal or ill-advised incision.

(d) *Clinical picture*

There is no sex preponderance. The cysts usually appear after puberty, but have been reported in infancy and in old age. The majority are first noticed



FIG. 98.—Thyroglossal cyst.

between the ages of 20 and 25 years. The infrahyoid variety tend to be slightly more common than the suprahyoid. If suprahyoid, the cysts are more common in the female sex. The cysts are usually fluctuant and rarely may be transilluminated (Fig. 98).

e) Differential diagnosis

Adenomas of the isthmus and solid tumours of the pyramidal process of the thyroid gland may be difficult to distinguish from these cysts, but in general they are firmer, non-transilluminable and do not move on protrusion of the tongue. An enlarged thyrohyoid bursa may cause a swelling which cannot be distinguished from a thyroglossal cyst until microscopical examination of the lining membrane reveals a single layer of thinned-out epithelium.

(f) Treatment

These cysts tend to grow larger and should be removed. It is, however, not sufficient to remove the cyst alone. To prevent the formation of a fistula the whole thyroglossal tract must be extirpated. Injection of dyes may help to map out the course of the tract up to the tongue and should always be attempted. It is not, however, always successful. If it is found at operation that the tumour is solid, the presence or absence of a normal thyroid should be determined, as the tumour may be the only thyroid tissue in the body. Its removal, if in the lingual or the suprahyoid situation, will still be necessary in such circumstances, but thyroid extract must be administered post-operatively.

(g) Operative technique

With the head extended, a transverse incision is made over the cyst and the median raphe between the sternohyoid muscles divided. The cyst is dissected out carefully and may be found to be attached to a track which extends upwards into the region of the hyoid bone. The fascia and muscle fibres surrounding this track should be removed in one piece with the sinus itself. On reaching the hyoid, its central portion is excised with bone forceps or strong scissors. The next step is to place the left index finger inside the mouth, and press with it upon the region of the foramen caecum in order to force the base of the tongue forwards and upwards. This manoeuvre is aided by traction on the tongue by means of a suture. A line bisecting the angle formed by horizontal and vertical lines below the mid-point of the hyoid is then followed by coring out all the tissues of the muscles of the tongue in a cylinder of about 3 millimetres in diameter (Fig. 99). This is best done with a tenotomy knife. The dissection should stop just short of the mucous membrane of the tongue. The gap in the tongue muscles is then closed with catgut sutures, care being taken to avoid the hypoglossal nerve. The defect in the hyoid bone may be left or the parts approximated with one or two sutures and the wound closed with drainage for 48 hours.

Incomplete removal will often lead to the formation of a fistula, and patients may give the history of three or four attempts at removal. If the excision is

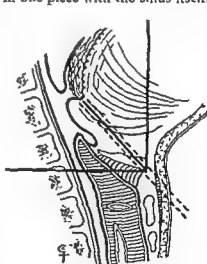


FIG. 99—Diagram illustrating the operation for removal of thyroglossal tract.

Stages of operation

Fistula formation

really radical as described above, results are very satisfactory. If the cyst is lingual it should be removed orally by blunt dissection.

3. LATERAL DEVELOPMENTAL CYSTS

(1) Branchial cyst

(a) Aetiology

Branchial remnants

This, as its name implies, is a cyst formed by inclusion of branchial remnants. At the end of the fifth week of intra-uterine life the third or fourth arches come to lie at the bottom of a depression—the pre-cervical sinus. A branchial cyst is formed either from the pre-cervical sinus, in which case it is lined with squamous epithelium, or, more rarely, from the second or third clefts, when it is lined by columnar epithelium which is occasionally ciliated.

(b) Surgical anatomy

Branchial cysts are usually found in the area between the sternomastoid muscle and the thyroid and hyoid cartilages, but may sometimes be seen higher up behind the angle of the mandible. In every case they emerge from under the sternomastoid muscle.

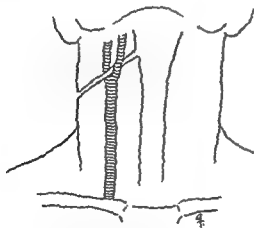


FIG. 100.—Diagrammatic representation of the track of a branchial fistula.

Deeply they are in relation to the great vessels and, if connected to a track, extend down to the pharynx between the internal and external carotid arteries (Fig. 100). They are often adherent to the internal jugular vein.

(c) Pathology

The cyst, if superficial, is filled with sebaceous material which is much more fluid than the contents of a dermoid or a sebaceous cyst. The constituents of this fluid are epithelial cells, fat and cholesterol. The lining membrane is a

squamous epithelium. If situated deeper the cyst may be lined by columnar cells and sometimes ciliated epithelium, and the contents are mucoid. This latter variety is, however, very rare and does not often cause symptoms. There is much lymphatic tissue around the lining of the cyst. A rare malignant tumour—a branchiogenic carcinoma—may originate in branchial rests. Its origin from a cyst is very uncommon.

Branchiogenic carcinoma

(d) Clinical picture

These cysts grow slowly and painlessly. Like thyroglossal cysts, they are usually first noticed in adolescence, and like them may on occasions be present at birth, or appear for the first time in old age. They form an oval swelling placed obliquely in the neck. They are soft and smooth, fluctuant but not transilluminable. They do not move on swallowing. Active contraction of the sternomastoid muscle will show that the muscle overlies part of the cyst. They

vary in size from time to time and may become secondarily infected (Fig. 101). There is said to be a familial incidence, transmission being through the mother, but they are commoner in males than in females. They may rupture

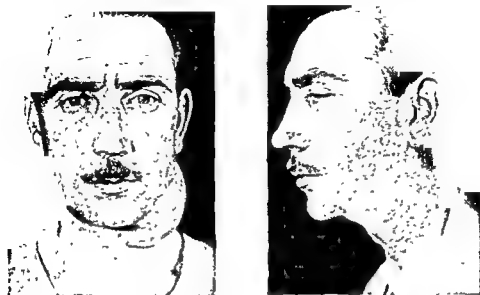


FIG. 101.—Large branchial cyst.

spontaneously after infection or may be opened in error. In both events a branchial sinus will form.

(e) *Differential diagnosis*

The cyst may be aspirated. The aspirated fluid closely resembles thin tuberculous pus at first sight, but it may show the shimmer of cholesterol crystals, which will be evident on microscopy. If after aspiration a fluid opaque to x-rays is injected, a definite outline of the cyst will be shown radiographically.

*Radio-
diagnosis*

A tuberculous abscess, whether from spinal caries or from glands which are breaking down, simulates a branchial cyst closely. Enlarged cervical glands may, however, be felt or spinal disease demonstrated. An added difficulty is that the cyst contents may occasionally contain a few tubercle bacilli. The aspiration replacement test described above will, however, in the case of a tuberculous abscess, not show the definite outline of a branchial cyst.

Diagnosis from a lipoma or a cyst in one of the thyroid lobes is much easier. Cystic hygroma (transilluminable), a solitary lymph cyst and haemangioma are other tumours from which branchial cysts must be distinguished.

(f) *Treatment*

The treatment is surgical removal. The cyst is exposed by a transverse incision over the lump or by an oblique incision in the line of the skin creases. The swelling is easily dissected out, but care must be taken in freeing its deep aspect as the cyst dips down in close relationship with the carotid arteries and may track up between them to the suprasternal fossa. As with

(2) Cystic hygroma

This is a rare, benign tumour which in about half the cases is present at birth and in about 90 per cent of cases is present by the end of the first year.

(a) Aetiology

The earliest formation of the lymphatic system begins in two large jugular sacs near the junction of the internal jugular and subclavian veins. Defective development, coupled with failure to unite with the vein, gives rise to a new growth composed of lymphatics, which appears as a soft, compressible tumour at the base of the neck and is known as a cystic hygroma (Fig. 102).



FIG. 102.—Cystic hygroma.

(b) Surgical anatomy

These cysts are diffuse and large and lie low down in the antero-lateral part of the neck. Their anatomical relationships are hard to define; they spread deeply below the sternomastoid muscle and often extend over the chest wall into the axilla or even into the mediastinum.

(c) Pathology

The cysts are multilocular, thin-walled and are composed microscopically of spaces lined by endothelium or simple connective tissue which contains a thin serous

fluid and some cholesterol crystals. They are, in essence, simple tumours of lymphatic tissue. They are subject to attacks of inflammation which lead to greater or lesser degrees of fibrosis.

(d) Clinical picture

The tumour is usually large. It is painless but causes great deformity. Its outline is regular and the tumour is very soft and elastic to the touch. It is translucent and the overlying skin to which it is attached is often pearly-blue in colour. The cyst expands when the child coughs or cries. There are, as a rule, no symptoms but, if the cyst is very large, some pressure on the trachea may be caused.

(e) Aids to diagnosis

Duffy's test is useful in these cases. He aspirates 20 cubic centimetres of fluid from the cyst and without removing the needle injects an equal amount of 15 per cent sodium iodide. A skiagram taken immediately after injection of the dye shows the extent of the tumour and is useful if surgical excision is contemplated.

(f) *Differential diagnosis*

This is from branchial cysts, lipomas and cavernous haemangioma.

(g) *Treatment*

The following methods of treatment are advocated.

(1) Surgical excision. This should not be contemplated until the child is over a year old. It may be difficult or even impossible to remove the whole tumour, but excision of most of the mass produces good results.

(2) X-ray therapy. This may cause a complete regression in many cases, and in others will produce shrinkage which will make radical surgical excision a possibility.

Surgical excision would appear to be the best treatment for small cysts and combined x-ray irradiation and surgery for large, extensive cysts.

(3) *Solitary lymphatic cyst*

A hydrocele of the neck is a large, smooth, unilocular cyst, thin-walled and tense. It lies posterior to the sternomastoid muscle in the lower anterior part of the neck. It appears in adult life and its origin is obscure. It is probably due to sequestration of the primitive jugular lymph sac. It can be dissected out with comparative ease. These cysts must be distinguished from a pneumatocele or hernia of the lung, which forms a resonant tumour in the posterior triangle of the neck above the clavicle; such a swelling distends on coughing, and breath sounds can be heard on auscultation.

(4) *Blood cysts*

Blood cysts are due to congenital herniation of the large veins of the neck which may become completely separated from the parent vessel, or may communicate with it by a very small channel. They should be carefully dissected out and removed after ligation of their attachment to the vein.

Occasionally blood cysts are caused by breakdown of a cavernous haemangioma. These should be approached with great care, as the haemangioma may spread widely amongst the muscles and connective tissues of the neck.

BIBLIOGRAPHY

- Bailey, H (1933). *Brit J Surg.*, 21, 173
 Christopher, F. (1924). *Surg Gynec Obstet.*, 38, 329.
 Duffy, J. J. (1937) *J Iowa St med Soc*, 27, 205.
 Gross, R. E., and Goeringer, C. F. (1939) *Surg. Gynec. Obstet.*, 69, 48.
 Joll, C. A. (1932) *Diseases of the Thyroid Gland, with Special Reference to Thyrotoxicosis* London; Heinemann.
 Sistrunk, W. E. (1920) *Ann. Surg.*, 71, 121
 — (1928). *Surg. Gynec. Obstet.*, 46, 109.

[References to other titles are given under Neck—Cystic Swellings of, in the Index Volume. The subject is also dealt with under the heading of Neck: Tumours and other Morbid Conditions, in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 104.]

NECK—TUBERCULOUS GLANDS

By RONALD W. REID, M.S., F.R.C.S.

SURGEON, ESSEX COUNTY HOSPITAL, COLCHESTER; VISITING SURGEON,
BLACK NOTLEY SANATORIUM, ESSEX; CONSULTING SURGEON, ESSEX COUNTY
COUNCIL

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1. DEFINITION

239.] Tuberculosis of the glands in the neck is the outward sign that tubercle bacilli have successfully invaded the lymphatic tissues which guard the upper part of the alimentary and respiratory tracts. Tuberculosis, the most lethal single disease affecting mankind, commonly makes its first assault upon the lymphatic and endothelial systems which are particularly active and important in the young. *Scrofula*, the old name for surgical tuberculosis, of which this disease is a common and striking variety, was clearly described by Richard Wiseman in the seventeenth century, when as "King's Evil" it was believed to be curable by the "Royal Touch".

2. AETIOLOGY AND PATHOLOGY

The tubercle bacillus, as Osler stated, is ubiquitous and is capable of long survival outside the human body. The chief external sources of infection are phthisical patients and the milk from tuberculous cows. The two common forms of bacillus, human and bovine, both cause tuberculosis of the glands in the neck. In urban dwellers the human bacillus is more common, whereas in rural patients the bovine bacillus occurs more frequently (Blacklock, 1947). These findings emphasize the need for public health measures to eliminate the sources of infection. *Sources of infection*

The tubercle bacillus possesses a remarkable power of penetrating the mucous membranes; it is probable that the acute catarrhal infections of childhood provide the site of lessened resistance through which bacilli gain entrance. Wherever they lodge, tubercle bacilli cause a local reaction which is cellular and exudative. Lymphatic and endothelial cells predominate in the cellular reactions, and form the typical tubercle from which the disease gets its name. The exudative response, allergic in nature, is particularly florid in the young. *Local reaction*

The evolution of tuberculosis depends finally upon the natural resistance of the patient and the virulence and dose of the invading bacilli. The bacilli may be exterminated at the point of entry, but more often they gain access to the lymphatic channels and reach the regional lymph glands. Here again there is an intense cellular and exudative response, so great in some cases that gross tissue death occurs with caseous suppuration. The reaction at the point of entry, which is the tonsil or other lymphatic mass in the pharynx, and the resistance in the regional lymph glands, constitute the primary tuberculous complex in the neck. *Natural resistance*

As natural resistance increases and local reaction diminishes, the early florid exudate absorbs and a deep cellular barrier is laid down round the bacilli and the tissues destroyed by them. The caseous pus may be absorbed, discharged to the body surface or, in the end, impregnated with calcium salts.

3. SURGICAL ANATOMY

The mouth and pharynx, the expanded upper parts of the alimentary and respiratory tracts, are backed by an extensive lymphatic system. Though widely distributed under the mucous membrane, the lymphatic tissue exists also in main masses—the pharyngeal and palatine tonsils. From the mucosa and from the lymphoid masses, a rich network of channels drain into the deep cervical lymph glands which are disposed in chains along the internal jugular vein. Into these glands drain also the superficial lymphatic vessels and glands of the scalp, face, neck and mouth, and at its lower end the deep cervical chain is in communication with glands in the upper mediastinum. The deep cervical chain is divided into superior and inferior parts. The superior is subdivided into two groups, medial and lateral to the internal jugular vein. The medial group lies in close contact with the postero-lateral aspect of the pharyngeal wall; one gland lying at the lower edge of the posterior belly of the digastric muscle has been called the tonsillar gland from the frequency with which it enlarges in tonsillar infections. The lateral group, behind and outside the vein, extends outwards towards the posterior triangle under the *Lymphatic system*

Age and
sex
incidence

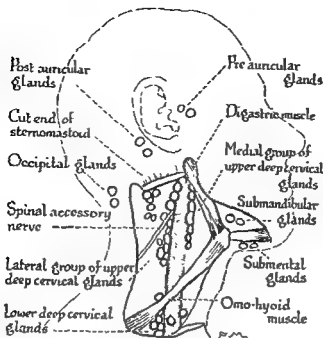


FIG. 103.—Semi-diagrammatic drawing of the deep planes of the neck with the sternomastoid muscle removed, showing the jugular vein and cervical lymph glands.

sternomastoid muscle and round the spinal accessory nerve. The efferents from the upper deep cervical glands pass to the lower chain, and so to the main venous system together with the lymphatics to the mediastinum and upper limb.

4 CLINICAL PICTURE

Tuberculosis of the glands in the neck is a disease of childhood, but adults and the aged are not immune and both sexes are equally affected. The disease occurs in three clinical forms: (1) local caseous tuberculous lymphadenitis of the upper cervical

chain, (2) caseous tuberculous adenitis of the lower cervical chain, and (3) diffuse non-caseating tuberculous lymphadenitis.

(1) Caseous tuberculous lymphadenitis of upper cervical chain

Caseous tuberculous lymphadenitis of the upper cervical chain is the common form; it is usually unilateral and is part of the primary complex of tuberculosis. The onset may be either gradual or sudden and may follow an acute specific fever or a respiratory infection. Often there is associated malaise with slight evening temperature. In over half the cases the tonsils are enlarged or otherwise abnormal and many show microscopical evidence of tuberculosis. This form of the disease may be seen in three stages, which are phases in the one continuous process ending in natural repair.

Stage 1.—This is the acute phase of invasion distinguished by soft, gross enlargement of the glands and peri-adenitis, a widespread oedema which mats together the glands and all surrounding anatomical structures. Suppuration is common, being gross when reaction is intense; it is confined at first to the glands, but soon breaks out and penetrates the fascia and the platysma to form a subcutaneous swelling—the superficial locus of the characteristic collar-stud abscess. The exudative response causing peri-adenitis is at first due to tuberculosis, but at any stage secondary infection from the pharynx or through attenuated skin may complicate and intensify the reaction and add to tissue destruction.

Stage 2.—As natural resistance increases, peri-adenitis diminishes and *pari passu* the glands become more discrete, firm, palpable and movable. The process is assisted by the discharge of tuberculous pus or by the elimination of secondary infection.

Onset

Suppuration

Collar-stud
abscess

Stage 3.—The third stage, when all peri-adenitis has disappeared and those glands in which actual necrosis has not occurred have returned to normal, is characterized by the presence of one or a few caseating glands rapidly going forward to calcification, the final victory of natural resistance. Such calcified glands may discharge their contents on the surface, but usually they remain as stony nodes in an otherwise normal neck. *Calcification*

(2) Caseous tuberculous lymphadenitis of lower cervical chain

In childhood caseous tuberculous lymphadenitis of the lower cervical chain is less common. The glands form a tumour, often with an abscess or a sinus in the supraclavicular region deep under the lower end of the sternomastoid muscle, and are simply the upper extension of intrathoracic tuberculous lymphadenitis. Search must always be made for the underlying lesions in the chest or even in the abdomen.

(3) Diffuse non-caseating tuberculous lymphadenitis

Diffuse non-caseating tuberculous lymphadenitis is a disease distinct from, and far less common than, the caseous form. Although it occurs at all ages, it is usually seen in children and young adults. The characteristic feature is a widespread, bilateral, rubbery enlargement of cervical lymph glands, and, on occasion, of many other groups—notably the axillary group. The glands remain discrete and movable although occasionally several may be matted together to form a rubbery tumour. General symptoms are not severe and the progress of the disease is slow. Treatment by constitutional measures has less influence on this type of tuberculosis than on the caseous variety, and the excision of glands, even if persisted in through multiple operations, is followed by recurrence. These enlarged glands are easily confused with those of lymphadenoma, a disease which may be complicated by tuberculosis. The aetiology of this form of tuberculosis is obscure. It may be that the infection is blood-borne, but there is little evidence to show how the bacillus gains an entry and why the reaction it produces should be so different from that seen in the primary complex. *Characteristic feature*

5. SPECIAL AIDS TO DIAGNOSIS

If an abscess or a sinus is present with tuberculous glands in the neck the diagnosis is usually clear, but beforehand the following tests may assist.

(1) Cutaneous sensitivity tests

The Mantoux and von Pirquet cutaneous sensitivity tests become positive soon after infection with *B. tuberculosis*.

(2) Aspiration

Aspiration and examination of the pus directly by culture or by guinea-pig test may occasionally be necessary, but care should be taken to avoid secondary infection or sinus formation.

(3) Biopsy

Biopsy of the glands should be avoided if possible, but it may be necessary to distinguish diffuse non-caseating tuberculous adenitis.

(4) X-ray examination

X-ray examination of the chest and of other suspicious areas should never be omitted.

6. DIFFERENTIAL DIAGNOSIS

The first step in the differential diagnosis of tuberculous glands in the neck is the recognition of whether the swelling is one of lymph glands or otherwise.

(1) Swellings of lymphatic glands

(a) Inflammatory

Distinguishing symptoms

(i) *Acute septic lymphadenitis*.—This is distinguished by the rapid onset, the brawny hot swelling, the high temperature, the severe malaise and the presence in the lymphatic drainage area of an acute infective lesion.

Primary lesions

(ii) *Chronic septic lymphadenitis*.—Chronic septic lymphadenitis presents a much more difficult problem. Search must be made in the drainage area for the primary lesion, including dermatitis of the scalp or pediculosis.

(iii) *Syphilis*.—In its primary form, syphilis gives rise to swelling of the glands and a search must be made of the lips and mouth for a chancre.

(b) Neoplastic diseases

Primary tumours

(i) *Carcinoma*.—Secondary carcinoma and branchiogenic carcinoma in lymph glands give rise to hard swellings which are at first discrete, and are encountered at an age when glandular tuberculosis is uncommon. If clinical grounds give rise to suspicion, a search for primary tumours in the mouth, pharynx, thyroid gland and chest must be made by general and special measures.

Hodgkin's disease

(ii) *Reticuloses*.—The reticuloses are new growths arising anywhere in the reticulo-endothelial system, including the lymph glands. They occur usually in adult life and give rise to a gradual, discrete and rubbery swelling of one group of lymph glands. This condition, which includes lymphosarcoma, can give rise to the greatest difficulty in differential diagnosis, and biopsy may be necessary.

Hodgkin's disease causes a rubbery enlargement of lymphatic glands, discrete at first, and followed by enlargement of the spleen, the Pel-Ebstein temperature and eosinophilia.

Paul-Bunnell test

(c) *General disease associated with chronic enlargement of lymphatic glands*
Glandular fever.—Glandular fever occurs in childhood and is a febrile disease associated with enlargement of the neck glands. The diagnosis may be made by the Paul-Bunnell test.

(2) Swellings other than in lymphatic glands

(a) Branchial cyst

Branchial cyst is a rounded, fluctuant, cold swelling in the upper part of the neck near the angle of the jaw. It increases slowly in size, is detached from the skin and is translucent to light. Unless infection occurs, the stigmata of inflammation are absent.

(b) Swellings of thyroid gland

Swellings of the thyroid gland are situated low in the neck near the midline and move with the larynx, though carcinoma arising in aberrant thyroid tissue may appear as a chain or a cluster of gland-like swellings in the posterior triangle of the neck.

(c) Parotid and submandibular swellings

These may be inflammatory, associated with calculus or due to new growth and may cause confusion by their resemblance to tuberculous lymphadenitis.

In inflammation and calculus the swelling increases at meal-times, when the gland is active.

(d) *Actinomycosis*

Arising in the bone or in other tissue of the neck, actinomycosis causes a Multiple brawny swelling with multiple sinuses, the discharge from which contains sinuses sulphur granules of the ray fungus.

(e) *Cystic hygroma—lymphangioma*

Lymphangiomas, which may contain large cysts (cystic hygroma), appear most commonly in childhood as soft, translucent, ill-defined swellings, either at the base of the neck extending towards the axilla, or in the submandibular region extending into the floor of the mouth.

7. TREATMENT

In the treatment of tuberculous glands in the neck, as with all other forms of tuberculosis, it is essential to look upon the patient as a whole and to remember that all that medicine and surgery can do is to assist him to overcome his infection by constitutional treatment aided by certain technical procedures. Neglect of constitutional treatment and ill-timed operations spell disaster, especially in childhood when general resistance may be low, as, for example, after a specific fever. *Constitutional treatment*

As soon as tuberculosis has been diagnosed the patient should be put at rest in bed, if possible in a sanatorium or a hospital equipped for open-air treatment. Failing this, home conditions should be made to approach as nearly as possible to the ideal. Rest in the open air, a liberal, interesting and nutritious diet, sunlight and vitamin therapy are essential, and should be continued until there is clear evidence that the patient's general resistance has been raised to a high pitch. In severe cases, local rest to the neck assists resolution. This can be procured most conveniently by means of a plaster collar which includes the head and shoulders, or by a splint constructed of strip metal and straps. *Local rest to neck*

The response to constitutional treatment is usually evident in from 6 to 12 weeks. The general health improves, the erythrocyte sedimentation rate falls and peri-adenitis subsides. The enlarged glands diminish in number and size and become more discrete. Resolution is often assisted by the removal of enlarged tonsils and adenoids and attention to sources of infection in the mouth, by the evacuation of a cold abscess and the treatment of secondary infection. *Removal of sources of infection*

Caseous tuberculous adenitis at the base of the neck and the widespread non-caseating form are less responsive to all forms of treatment, including constitutional measures and surgical excision, than is the common localized type seen in the upper deep cervical glands. Sanatorium treatment must therefore be thorough, and should be related to the extent of intrathoracic disease if such is present. In some recalcitrant cases radium or deep x-ray therapy may assist in procuring resolution or fibrosis of the affected glands. *Radium and x-ray therapy*

8. INDICATIONS FOR SURGICAL INTERVENTION

(1) Cold abscess

Aspiration is less satisfactory than incision, curettage and suture of the wound. This minor operation should be performed without hesitation in all

cases with clinically obvious pus, for thereby resolution is assisted and secondary infection is avoided.

(2) Secondary infection

This complication should be treated by free incision and drainage of any accumulations of pus found. Thorough chemotherapy, including the use of penicillin, is indicated. Secondary infection adds to the seriousness of the disease and therefore should be avoided at all costs.

Penicillin

(3) Excision of tuberculous glands

This is indicated only when full constitutional treatment has brought about the maximal resolution, as shown by improvement in the general condition and the disappearance of peri-adenitis. Excision should not be attempted even if glands threaten to break down despite constitutional measures. In such circumstances there will be much matting of glands by peri-adenitis, anatomical structures will be obscured and tuberculous pus encountered. Incision and curettage would be the correct treatment. Operations for the removal of glands in the base of the neck should be undertaken only when there is evidence that the intrathoracic source of disease is under therapeutic control. In this form and in diffuse tuberculous lymphadenitis, disappointment inevitably follows operations which are conducted early in the clinical course of the disease, for recurrence is the rule.

Glands at base of neck

9. OPERATIVE TECHNIQUE

Unless there are strong contra-indications, anaesthesia should be general and given by means of an endotracheal tube. Thus a clear and controlled airway is assured and the operation field remains undisturbed. A combination of gas, oxygen and ether is very satisfactory.

Anaesthesia

The skin having been prepared, the patient's head is turned to the sound side, and a large flat sand-bag so placed beneath that the affected area is thrown into prominence. A natural transverse crease in the neck over the tumour is chosen for the incision, which should include any sinus or diseased skin. More than one transverse incision may be required for the excision of many glands. Transverse incisions are much preferable to a vertical cut which invariably gives a poor cosmetic result. The skin and the platysma are reflected together off the mass and a self-retaining mastoid retractor is inserted. If a sinus is present, dissection may be simplified by inserting a director and pulling up the gland. The sternomastoid muscle is then dissected backwards off the mass, if it is of the upper deep cervical chain; the fascia in front is picked up and, by dissection close to the glands, a line of cleavage is sought. By gauze and scissors the glands are cleared in front; the internal jugular vein is usually found to be adherent but easily detachable. Behind and under the sternomastoid muscle careful search should be made for the spinal accessory nerve, which is preserved, if necessary, by splitting the gland mass. Plugs of gauze should be inserted alongside the cleared glands to control minor haemorrhage. The lowest gland affected should now be sought and the whole mass turned up towards the posterior belly of the digastric muscle, which must be carefully cleared until the uppermost gland, usually by the transverse process of the axis, can be raised up leaving a pedicle to be

Transverse incisions

clamped and tied. The facial and lingual arteries and the hypoglossal nerve should be avoided.

All enlarged, firm, or caseous glands must be removed, but soft seedling glands may be left behind. If, in any part of the operation, the gland mass is found to be ill-defined and to involve important structures, injury of which would be prejudicial to the patient, it is best to avoid risk of injury by curetting the affected part, for small residual areas of caseous tissue are not likely to prevent healing by first intention, or to cause recurrence of disease if sufficient constitutional treatment has been given.

When haemostasis has been secured the deep space is closed as far as possible by fine deep sutures, the platysma is united with the same material and the skin closed with ophthalmic salmon-gut. A drain inserted for 24 hours in the posterior part of the wound is useful in preventing a haematoma, which may organize if any residual space is left. Stitches are loosely tied and are removed in 4 days; if this is done and the platysma is properly sutured, the cosmetic results should be excellent. *Drainage*

10. PROGNOSIS

The prognosis of tuberculous glands in the neck is good so far as danger to life is concerned, but account must be taken of the glands affected. In the common form of the disease in which the bacilli have invaded the nasopharynx and have been fixed in the upper cervical lymph glands, a breakthrough to the blood stream and dissemination are practically unknown. This lesion, a primary complex, responds favourably to constitutional treatment and is amenable, when need be, to surgical extirpation. *Constitutional treatment*

Tuberculous lymphadenitis of the lower cervical glands means visceral disease in the thorax, and the prognosis depends upon the nature and extent of that lesion. Constitutional measures remain the sheet anchor of treatment, for excision of glands can never be other than incomplete.

Diffuse tuberculous lymphadenitis is slow to respond to constitutional treatment, and excision—no matter how painstaking—is almost always followed by recurrence. The course of the disease is long and treatment calls for great patience, but the outlook for resolution is good.

Provided that general principles are kept in mind and that adequate preliminary constitutional and local treatment precede surgical excision, recurrence of the common form of tuberculous glands in the neck is unusual and the cosmetic results are good. In a series of cases quoted by Wilkinson (1942) satisfactory results were obtained in 90 per cent of cases treated by constitutional measures combined with formal excision, recurrence was noted in 7 per cent and no patient developed systemic tuberculous lesions. *Good result by combined methods*

REFERENCES

- Blacklock, J. W. S. (1947). *Brit. med. J.*, **1**, 707.
 Wilkinson, M. C. (1942). *Non-Pulmonary Tuberculosis*, p. 29. London; Hamish Hamilton

[References to other titles are given under Neck—Tuberculous Glands, in the Index Volume.]

NEOPLASMS—INNOCENT AND MALIGNANT

By J. HENRY DIBLE, M.B., F.R.C.P.

PROFESSOR OF PATHOLOGY, BRITISH POSTGRADUATE MEDICAL SCHOOL,
UNIVERSITY OF LONDON

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1. NORMAL AND PATHOLOGICAL GROWTH

240.] Although it is customary to prefix to a study of neoplasia a consideration of normal growth, there are fundamental differences between the two and also between neoplasia and overgrowth. The latter may be the result either of the enlargement of the cells of an organ—of which the hypertrophy of the fibres of the pregnant uterus, or of the cardiac muscle in response to a call for extra work, are familiar examples—or it may be due to an increase in the number of the constituent cells; for example, the hyperplasia of red or white marrow cells under the stimulus of anoxia or of infection. Both these processes of overgrowth are a response to a recognizable bodily need, and in each case there is a return to what may be called normal limits if the stimulus causing the overgrowth is removed. Neoplasia comes into quite a different category. In the first place it is purposeless, for whatever the stimulus may be which sets the process going there is no evidence that it is of value to the body, either in the performance of normal or of increased function, or as a defence measure against invasion by bacillus or virus. Secondly, this type of overgrowth always takes the form of cell multiplication; there is no such thing as neoplastic hypertrophy.

(1) Neoplasia and function

It is no contradiction of the generalization that tumours are without function in the economy of the body to recall that certain of them produce organized tissue. Thus we may progress in sequence from the spindle-cell sarcoma, with little intercellular material, to the fibrosarcoma with a pronounced formation of collagen fibres; and in like manner ossein or cartilage may be produced by both malignant tumours and simple growths.

(2) Recognition of tumour types

This process of hyperplasia and differentiation is related to the length of the life-history of the cells which constitute the tumour, and it is upon our recognition of it that the classification of tumours mainly depends. The simplest types—carcinoma and sarcoma simplex—are merely masses of embryonic undifferentiated epithelial or connective-tissue cells, and their status in histological diagnosis depends upon the recognition of the type of tissue to which such a structure conforms. The epithelial cell has no ability to form its own vascular supply, and is always dependent upon a connective-tissue base or framework for its support, and to carry the vessels necessary for the nourishment and continued growth of its cells; it is the recognition of these features which enables us to diagnose such a tumour as a carcinoma. The growth of connective tissue, which proceeds more or less *pari passu* with that of the malignant epithelial cells, is known as the stroma reaction and is usually regarded as secondary to the epithelial growth and is itself non-malignant in character. It is, however, a matter worthy of consideration that it is upon this response that the existence of the tumour depends.

The degree of stromal reaction varies greatly from tumour to tumour and its presence or absence largely determines the physical characters of the growth. Thus a relative preponderance of epithelial cells, which form large masses with little supporting stroma, will produce a soft and friable growth, with a tendency to necrosis because of the distance which the cells may be from the stromal blood-vessels. On the other hand, a pronounced stromal reaction, with relatively little epithelium, will lead to the production of a hard and fibrous tumour, the common example of which is the "scirrhous" of the breast. It is sometimes suggested that the fibrous tissue in such growths has a protective function and may "strangle" the tumour cells—pictures of the scanty cancer cells in old collagenous fibrous tissue of the "atrophic scirrhous" being adduced to support this belief. It seems unlikely that such an interpretation is correct, and it is more probable that some unknown factor limits the rate of growth of the epithelial cells. An abundant stromal reaction is certainly no valid index of low malignancy.

(3) Differentiation as a process in tumours

It is not necessary, in a work of this type, to recapitulate the formal classifications of tumours, which are sufficiently well known. It may be stressed, however, that the recognition of subtypes of growth, within the great classes of epithelial and connective-tissue tumours, depends upon the recognition of adult forms of tissue produced by the growth of the cells (in innocent tumours), or of attempts at this which fall more or less short of completion (in malignant tumours). Thus in a fibroma, or myoma, well-developed collagenous fibrous tissue or muscle are produced, albeit arranged in an irregular and formless fashion, whereas in a carcinoma or sarcoma ill-formed glandular tubes or stratified epithelium in the one, and fibroblasts or osteoblasts in the other, enable us to classify the growth as an adenocarcinoma, a squamous-cell carcinoma, or a fibrosarcoma or osteosarcoma as the case may be.

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1. NORMAL AND PATHOLOGICAL GROWTH

Overgrowth

Neoplasia

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PROFESSOR OF PATHOLOGY, BRITISH POSTGRADUATE MEDICAL SCHOOL,
UNIVERSITY OF LONDON

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1. NORMAL AND PATHOLOGICAL GROWTH

Overgrowth

Neoplasia

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tumour cells from a simple undifferentiated embryonic form to a stage at which they present the characters of a recognizable tissue. This conception is necessary for histological diagnosis and, what is of equal importance, for a comprehension of the malignant behaviour of certain growths.

Put into rather different and general terms, it may be said that the degree of differentiation of a growth is a function of the length of the life-history of the cells composing it. If this is short the cells quickly reach the limited development allotted to them. If it is long the tumour grows slowly and the cells reach adult development and assume the form they have in adult tissue. In doing so they show some evidence of their normal function but in a local fashion. This is a trivial matter in tumours of connective-tissue or surface epithelium, but if endocrine cells are involved the production of an excess of powerful metabolic substances may produce very striking results; thus hypoglycaemia from pancreatic islet growths (nesidioblastoma), Cushing's syndrome from basophil pituitary growths, gigantism from growths of the acidophilic cells of the same organ, virilism from adrenal and ovarian growths (arrhenoma) and bone absorption from parathyroid growths may be instanced as a few examples of such a phenomenon. This function is useless for the bodily economy, but on the morphological side similar effects are seen in the production of collagen by fibroblasts or muscle by myoblasts, or of a collection of useless gland tubules in adenomas and so on. Not only does this formation of normal tissue demand time, so that such a tumour grows slowly, but the cellular arrangement is itself one which does not favour dissemination, the constituent neoplastic cells being anchored in position by the related stroma or by the intercellular connective-tissue structure which they produce. Such growths are described as tissue tumours or histiomas. By contrast, a typical malignant tumour is composed of cells which retain the undifferentiated form of undeveloped connective tissue, or of epithelial cells lacking the features and arrangement of mature epithelium. The life-history of such cells is short; that is, the time from mitotic division to the end of their very limited development is short, and hence the growth tends to be rapid. The tumours composed of such undifferentiated cells are known as cytomas and these form the class of malignant neoplasms.

(4) Latency in malignant tumours

One of the anomalies of malignant tumour growth which is out of keeping with the foregoing remarks is the extraordinary latency which is sometimes shown. It is well known that malignant growths may recur after many years and that the recurrent growth may proceed at least as fast as the original primary growth, if not faster. If the cells which produce the recurrence, and which it is assumed had been left behind at the time of operation, were to have continued their previous life-history at the site at which they had been left, it would seem that a recurrent growth must have appeared in a short time, but this may not be the case. For example, Turner (1937) has recorded a recurrence of a breast carcinoma in the axillary glands 25 years after a simple excision of the breast for malignant disease. The primary nodule of the tumour in the writer's case of synovioma (Fig. 104) was known to have been present for three weeks before it was excised. The recurrence took place 8 years later; there was no clinical evidence of any increased rate of growth, but

Endocrine
factors

Histioma

Cytoma

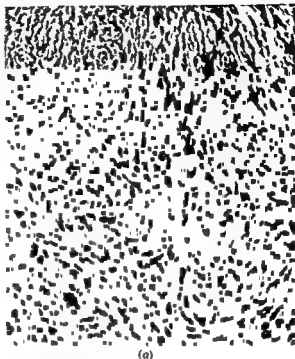
Recurrence
of malignant
growth

although amputation was performed the patient died from dissemination 3 months later. The melanoma of the eye is notorious in this respect, secondary growths in the liver, which prove rapidly fatal, appearing 8, 9 or even 15 years after the removal of a primary growth (Turner, 1939).

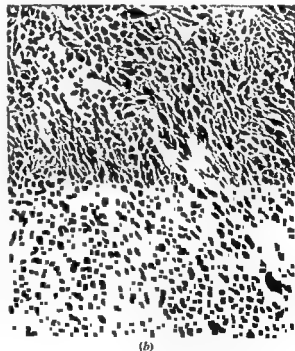
It would seem, therefore, that a state of latency is possible with some malignant cells, in which they remain in the tissues for long periods without dividing, or only divide occasionally. Later, after years of such dormancy, a stimulus is applied, a restraint removed, or an essential growth factor metabolized, and the cells suddenly proliferate again at their original rate.

(5) Effects of tumour cell differentiation

It is generally conceded that the differentiation of a cell beyond a certain stage involves the loss of the power of division. It is sometimes held that the process is reversible and that under stimulation such cells can "de-differentiate" and revert to a stage at which multiplication is again possible. Doubtless the state of affairs is not the same in all tissues. The liver cell, for example, seems to offer a case in which differentiation (in the sense of the elaboration of functional activity) does not connote loss of power to divide, for this cell, though structurally appearing simple, is one of the most versatile cells of the body from the



(a)

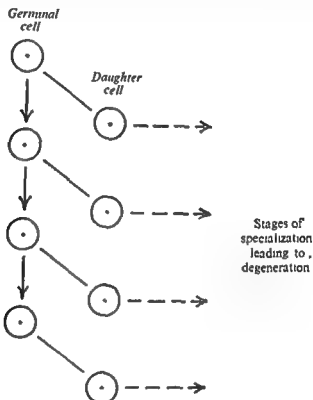


(b)

FIG. 104 —Fibrosarcoma ("synovioma") of elbow-joint capsule ($\times 160$) (a) Original growth, some weeks' duration, (b) Recurrence after eight years

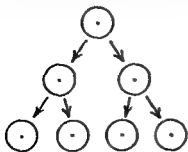
functional aspect, and from this angle may be considered highly differentiated. Nevertheless, its power of multiplication remains high and it can reproduce without any very obvious alteration in the cell's appearance. In most of the cells of the body, however, differentiation, when it takes the form of an elaboration of histological appearance, seems to connote loss of ability to reproduce. A good example is the skin. Here the process of multiplication takes place in the germinal layers and may be illustrated in a simplified form by the following diagram.

*Loss of
ability to
reproduce*



The daughter cells differentiate, mature, and are shed, therefore, at any period the number of germinal cells is roughly the same. The differentiating cells are embarked upon a path of development from which there is no regression. As they mature, die and are cast off, their loss is exactly balanced by production of new cells, and so the thickness of the skin is constant. If it were not so, and if the two daughter cells each possessed the power to subdivide, as shown in the diagram below, the skin would increase in thickness indefinitely.

*Balance of
cell
production*



Now there can be no doubt that a law of this kind governs the size and cellular content of all organs in which there is a turnover of cells. In hyperplasia, and in certain pathological conditions (such as leucocytosis), it is modified and, to take the last example, the size of the white marrow organ increases. This indicates that the condition is not merely an increased rate of multiplication, so that new cells are

produced and passed more quickly into the peripheral circulation, but that there is a fundamental alteration in balance within the marrow and a multiplication of daughter cells in excess of the normal; additional production lines (to use a current simile) are brought into being. With recovery and the removal of the stimulus to multiplication the marrow reverts to normal. This we must suppose involves a reversal of the process, and a degeneration of a certain number of the new germinal cells, so that the number of "production lines" is reduced. Whether this is or is not the sequence of events it appears that the formative process remains the same and results in the formation of differentiated cells which are used in the bodily economy. In tumour growth something of the same sort occurs, but there is an enormous preponderance of germinal cells in the malignant growths so that these form a bulky mass. Even here there is a degree of differentiation, and here also it appears that when once the cell has reached a certain stage of structural development it cannot revert to a more primitive state. We thus find no support for the doctrine of de-differentiation. Indeed, were it true, we should have the possibility that any form of tumour could arise in any tissue, since the reversion might well go back to the blastocyst stage of totipotent cells. Since we do not, in fact, find this—but in a general way all our experience is in the other direction—we are entitled to conclude that the proliferation of cells, both for physiological needs and neoplasia, is due to the constant presence in organs of specialized germinal cells of limited potentiality.

*Additional
production
lines*

*No reversion
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primitive
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Assuming that these facts are true, we conceive that the behaviour of a neoplasm depends upon the length of the life-history of its constituent cells. Is this constant for all the cells of the growth or does it vary from cell to cell? It appears to be a remarkable fact that it is relatively uniform for, as Ewing says, "a tumour process becomes established at a certain momentum which it tends to maintain throughout its entire course". The result is that most growths are definitely either benign or malignant. Moreover, the cells in a single growth appear to show very much the same rate of growth throughout the tumour. An exception to this general statement is the alteration in type of growth, which sometimes may occur spontaneously or sometimes follows incomplete surgical removal. When this happens the histological change (Fig. 104) is that the growth becomes more cellular and the cells less well differentiated and they possibly have a shorter life-history. This change from a benign to a malignant habit is often quoted, but apart from the melanomas in which irritation is known to cause the sudden appearance of malignant characters, it is probably less common than is taught. It is often assumed to characterize mixed parotid tumours, but McFarland (1942), in his masterly survey of the clinical course of these tumours (in which he estimates the incidence of recurrence to be of the order of 60 per cent and the average period between removal and recurrence to be about 7 years), barely considers this phenomenon, apart from rapid enlargement due to haemorrhage, necrosis, oedema, ulceration and suppuration which is not truly neoplastic.

*Uniformity
of behaviour*

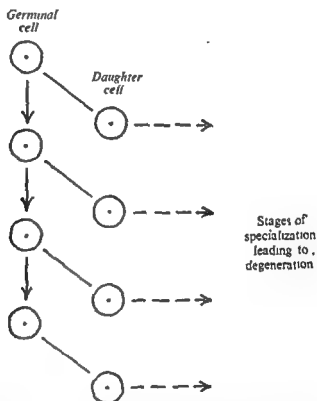
*The mixed
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tumour*

(6) Spread of tumours and considerations affecting their surgical treatment

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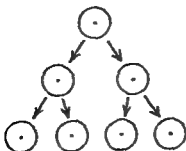
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and type of spread is related to the essential characters of the tumour—whether of epithelial or connective-tissue structure. Since epithelium is dependent upon stroma for its support and nutrition, epithelial cells can live only in a framework of fibrous tissue with its associated blood-vessels and lymphatics. A mass of epithelium cannot expand eccentrically, like an inflated balloon, except for a short period and at the expense of central necrosis. Eccentric expansion of an epithelial cancer, therefore, takes the form of the invasion of the tissue spaces, and this living injection forces its way into the communicating lymphatics just as a mechanical injection will do. The result is a local permeation of the lymphatics from which the following are the natural consequences. First, as the column of cells grows, a destruction of specialized tissue cells in the vicinity results, and a new formation of fibrous tissue stroma is brought into being. Secondly, epithelial cells may become detached and pass in the lymph stream to the related glands where, as foreign bodies, they tend to be arrested in the lymph sinuses, in which they continue their growth, destroying the special tissue of the gland and setting up the customary stroma reaction. There is little evidence, from histological examinations, that such emboli are ever destroyed in the glands, though it is conceivable that this may occur. Lymphatic spread of carcinoma is thus by two processes—permeation and embolism—and the relative importance of these is a matter of considerable disagreement. Handley (1922) has made out a good case for the extensive nature of permeation, more especially from studies of the breast and from post-mortem material, in which he has claimed that most of the distant deposits in this disease may be explained by this process, and he has shown long tracts of tissue containing lymphatic vessels stuffed with carcinoma cells. The occurrence of such a process is not denied, but issue has been joined concerning the period at which it takes place and its relative importance in promoting early metastatic deposits. The view more generally taken is that the early involvement of the lymph glands in such cases is due to embolism, and that the condition found by Handley is, in general, one of late development. The former, indeed, must often be the case, since the excision of a primary growth and its related lymph glands in areas in which a block dissection is not always possible, often gives good results in the presence of proved glandular metastases (Butlin, 1909). This could hardly happen were these glands involved by continuous permeation, unless the cancer cells retrogressed spontaneously in the intervening tissue—which seems unlikely. Nevertheless, the emphasis laid on lymphatic permeation has served a valuable purpose in establishing block dissection, with the removal of the primary growth, the related glands and the intervening tissue carrying the lymphatics, as the procedure *par excellence* in the surgical treatment of malignant epithelial tumours.

(7) Haematogenous extension

Blood-stream invasion by a carcinoma may take place locally in the primary tumour, or it may be delayed until the cancer has reached the lymph glands, in which they may extend for considerable distances by permeation or become detached and form emboli. The tendency of a carcinoma which has reached a gland is for it to destroy the proper tissues of the gland and to pursue its extension by the

Method of
expansion

Lymphatic
spread of
carcinoma

Early
involvement
of lymph
glands

Lymphatic
permeation

effluent lymphatics, either by permeation or embolism, or by both. The consequences of this are the involvement of more proximal glands, and, once the lymphatic current is obstructed, extension is possible to other peripheral glands by retrograde spread; ultimately extension by the lymphatic route leads to the thoracic duct and the blood stream. Once blood invasion has become established secondary growths may occur in the lungs (or in the liver in the portal zone) and either by the direct passage of unarrested tumour cells through the pulmonary capillaries, or by tertiary haematogenous spread into the pulmonary veins from secondary nodules in the lungs, blood-borne metastases may be produced anywhere in the area of the peripheral circulation.

(8) Sarcoma metastases

Sarcomas have little tendency to spread by lymphatics, if we exclude such growths as lymphosarcoma and reticulosarcoma, which originate in the

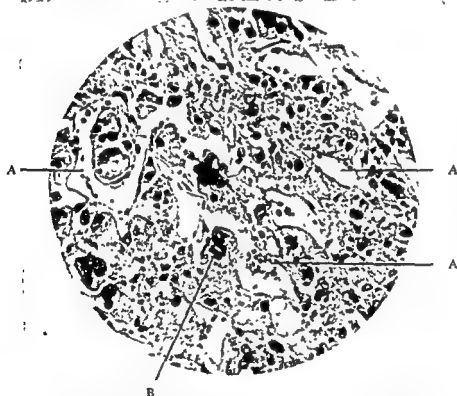


FIG. 105.—To show blood-vessels in a sarcoma A = blood spaces showing corpuscles and close juxtaposition of tumour cells B = tumour cell apparently free in blood space (liposarcoma).

lymphadenoid system and have essential and intimate relations with it, and malignant melanoma which may behave as a carcinoma. Again this character of the tumour's mode of dissemination results from the nature of the growths, which arouse no stromal reaction but are themselves essentially mesenchymatous and in their structure in many ways resemble granulation tissue. They are highly vascular and the blood-vessels upon which they depend are developed in tissue which is itself neoplastic. Hence there is ample

*Eccentric
growth of
sarcoma*

opportunity for tumour cells to enter the circulation either by amoeboid movement or as a result of trauma (Fig. 105). The experiments of J. S. Young (unpublished) upon models have indicated the extreme ease with which free cells in a gel-like medium may enter vessels which carry a current of fluid and which contain the smallest patency, under conditions of hydrostatic and tissue pressure such as may well obtain in tumours *in vivo*. The spread of a sarcoma by the lymphatics is certainly unusual and many typical growths of this type give the appearance of encapsulation, which means that their growth is eccentric rather than invasive and that circumjacent tissues are compressed rather than infiltrated; such an appearance, of course, has no bearing upon their malignancy, which arises not from their peripheral spread but from within the tumour by its capacity to invade the blood stream and to produce metastases by this route.

2. THE ASSESSMENT OF MALIGNANCY AND OF REACTION TO IRRADIATION

(1) *Histological assessment of malignancy*

This is one of the most important aspects of histological diagnosis and it requires the close collaboration of surgeon and pathologist if results of value are to be obtained.

The histological assessment of malignancy can only be the fruit of experience. It is only by co-ordinating the histological picture with the clinical course of the case that this prophetic skill can be acquired; long experience in the histology of tumours and full knowledge of the after-history of many cases are required. It may be said that few pathologists in the past have been in a position to obtain this experience, and that few surgeons have the histological skill to apply their clinical observations to this end. This state of affairs should be remedied.

Many attempts have been made to formulate rules, relating histological structure to malignancy, based upon the expected co-ordination between the degree of cell anaplasia and the rapidity of growth and the malignancy of a tumour. Broders (1920 and 1932), for example, classifies epithelioma into four groups, depending upon the differentiation of the cells present.

*Broders's
classification*

- Grade I* Three-quarters of the cells are differentiated
One-quarter are undifferentiated
Keratinized cell-nests numerous
- Grade II* Half of the cells are differentiated
Half are undifferentiated
A few cell-nests may be seen
- Grade III* One-quarter of the cells are differentiated
Three-quarters are undifferentiated
No cell-nests
- Grade IV* All the cells are undifferentiated

Broders states that he pays attention to the number of mitotic figures which are seen; when in doubt he tends to place the tumour into a more malignant group.

It is clear that many criticisms may be levelled against an arbitrary system of this sort, such as errors of sampling, personal factors, the question of site, the neglect of clinical considerations and so on. It might also have been a desirable simplification to have formulated three groups instead of four. Patey and Scarff (1928) have applied similar criteria to the assessment of the malignancy of breast cancers, but have paid regard to the ascertainable extent of the growth. Considering tubule formation, inequality in size of nuclei and hyperchromatism, the number of mitotic figures and evidence of secretion, they classified their growths into three groups, and in a small series of 50 cases submitted to radical operation by the same surgeon and followed up for from 3 to 7 years, they found some evidence of the usefulness of their classification in assessing the probable course of the disease—but this was overshadowed by the much greater significance of the presence or absence of axillary gland involvement. This, of course, is the paramount fact in the

*Malignancy
of breast
cancers*

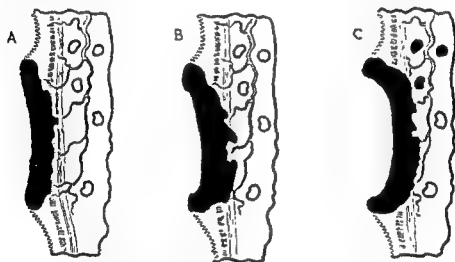


FIG. 106.—A = growth limited to wall of rectum B = extension of growth to extra-rectal tissues but no metastases in regional lymph nodes. C = metastases in regional lymph nodes (Dukes).

consideration of prognosis, and it may not be out of place here to refer to the careful studies of Dukes (1946) on carcinoma of the rectum. Using a modification of Broders's classification he divided 2,057 cases of cancer of the rectum into "low", "average" and "high" grades of malignancy and found that the percentage showing lymphatic metastases at the time of operation was 18 per cent in the low-malignancy group, 44 per cent in the average group and 80 per cent in the high-malignancy group. The supreme importance of the extent of spread and the existence of lymphatic metastases is also well shown by other observations of Dukes. He divided 716 cases, which were followed up for over 5 years, into the following groups (Fig. 106) Group A, in which the growth did not pass beyond the musculature Group B, in which the extra-rectal areolar adipose tissues were involved but the growth did not appear in the glands. Group C, in which the regional lymph glands were invaded. The following analysis of such cases, treated by radical excision, shows the relationship of the extent of spread to the post-operative progress. Of this

*Malignancy
of rectal
cancers*

large series only 12 patients were not followed up and these were assumed to have died of the disease.

GROUP	NO. OF PATIENTS SURVIVING OPERATION	ALIVE 5 YEARS AFTER OPERATION	PERCENTAGE OF PATIENTS SURVIVING FOR 5 YEARS
A	105 (14.7 per cent)	86	81.9
B	252 (35.2 per cent)	157	62.3
C	359 (50.1 per cent)	94	26.2

Unfortunately only 15 per cent of Dukes's cases fell into Group A, whereas 35 per cent were in Group B and 50 per cent in Group C.

(2) Diagnostic biopsy

This may be desirable for simple diagnosis or to control irradiation treatment. A first question is that of the possible risks to the patient of the procedure. It has been shown repeatedly in experiments upon animals that the manipulation of malignant tumours will cause the entry of cancer cells into the blood stream and increase the incidence of pulmonary metastases. Doubtless lymphatic emboli may be produced in the same way. Therefore it seems obvious that a grave responsibility rests upon the surgeon who allows the repeated and often unnecessarily violent manipulation of tumours by his students or even by his assistants. The writer recollects the admonition of Macewen, "We said 'palpate', sir, not 'massage'!" For the same reason the safety of the process of aspiration or puncture biopsy must, *a fortiori*, be viewed with grave doubts, as these procedures open up vessels and also increase the tissue pressure within the growth, creating conditions favourable to the passage of tumour cells into the circulation. (See Gland-puncture and Aspiration Biopsy, Vol. 4, p. 297.) The same objections do not seem to apply to "open" biopsy, which does not increase tissue pressure and in which bleeding may be assumed to tend to wash away tumour cells which are freed in the proceeding. The results of animal experiments (Willis, 1934; Ewing, 1940) support this conclusion, and Ewing states that many of the "5-year cures" in cases of osteogenic sarcoma recorded in the *American Registry of Bone Sarcoma* were subjected to biopsy examination. Biopsy, if resorted to, should be regarded as an important diagnostic procedure, and be carried out with care and, if possible, by someone experienced in the requirements of the pathologist. An adequate sample of the growth should be obtained, and this should include the growing edge of the tumour. A fragment of necrosing tissue from the surface is useless. Quite apart from the dangers of the procedure small haphazard biopsy fragments, such as may be obtained by the "punch" technique, although they may suffice for the diagnosis of the type of tumour, may be of little use if any assessment of its malignancy or of its response to irradiation be desired. The material obtained should be fixed in such a way as to afford a maximum of information: for the purpose "Susa" fixation can be recommended. "Susa" fluid is mercuric chloride, 45 grammes, sodium acid, for 3

Dangers of biopsy and of examinations

Method of biopsy

(3) Assessment of radiation effects

In deciding upon the treatment of malignant growths the surgeon is often faced with the difficult decision whether to advise surgical excision or radiotherapy, or a combination of both procedures. It is a question that sometimes does not admit of more than one solution, but there is a large number of cases in which a choice is open. At the present time such a decision has to be based upon personal experience (which is often deficient with regard to the alternative technique) and the evidence of statistics. Much information has now accumulated as to the radio-sensitivity of certain types of growth, and this can be correlated with a biopsy finding as soon as this is known, but clearly a great advance would be achieved if we were able, in each individual case, to test whether or not the response to irradiation was likely to be favourable. The recent work of Glücksmann (1941 and 1946) and of Glücksmann and Spear (1945) suggests that this may be practicable in the near future.

In considering the effect of irradiation it is important to distinguish between "radio-sensitivity" and "radio-curability". Attention in the past has been directed too much towards the former, and early experience with the immediate response of highly undifferentiated growths, such as an epithelioma in Broders's Group IV or a Ewing sarcoma of bone, suggested that such growths were more suitable for irradiation than those which contained many differentiated cells, such as an epithelioma in Broders's Group I. This view, based upon a speedy immediate reaction, has been found to be fallacious and we now recognize that "quick to go" too often connotes "quick to come back". It is one of the merits of Glücksmann and Spear's work that, by the analysis of the cellular response to irradiation from the examination of serial biopsies made upon tumours under treatment, they have established certain principles which permit an opinion of the radio-curability of tumours to be formulated. The original papers should be consulted, but broadly these principles are based upon the recognition and examination of the various types of cell in a

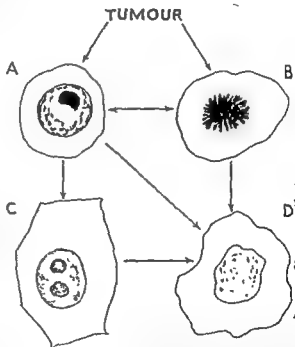


FIG. 107.—Diagram to show the constituent cells of a tumour. Viable cells: A = resting cell; B = mitotic cell. Non-viable cells: C = differentiating cell; D = degenerate cell. (Drawings from a squamous-cell carcinoma of the tongue)

Radio-sensitivity and radio-curability

*Types of
cell*

tumour population. This latter is made up of: (a) resting cells, which are actively growing cells capable of further division and of differentiation; (b) cells in active mitosis; (c) differentiating cells; and (d) degenerating cells. Only those in categories (a) and (b) are capable of further division and are the essential elements in the malignant process; those in categories (c) and (d) have passed beyond the stage of further multiplication and may be considered non-viable. Put in diagrammatic form the constituent cells of a tumour may be shown as in Fig. 107.

The diagram is, of course, an over-simplification, since the dividing cells may give rise to cells which divide again without differentiation, or they may differentiate, moreover cells at any phase of the cycle may degenerate.

Such an analysis informs us of the natural tendency of the tumour and gives in rather more detail the information vouchsafed by other systems which evaluate malignancy. When, however, the tumour is exposed to irradiation, and the same form of analysis is applied to a second biopsy and to later biopsies, it is claimed that information of prognostic value may be obtained by the enumeration of the above cell types. The effect of irradiation is to cause necrosis of cells and also to increase the tendency to cell differentiation, so that there is a general decline in the proportion of cells which are viable and can subsequently divide. By such means it is possible to follow the effect of irradiation and to say whether or not this method of treatment is likely to be effective.

Plate II shows examples of the application of this technique to epithelioma of the uterine cervix, which in one case reacts favourably to irradiation and in the second does not react.

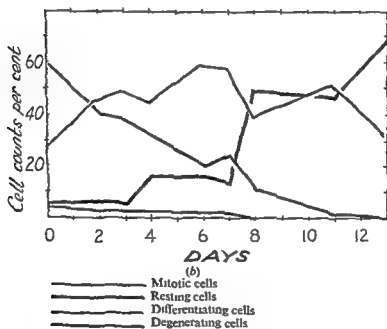
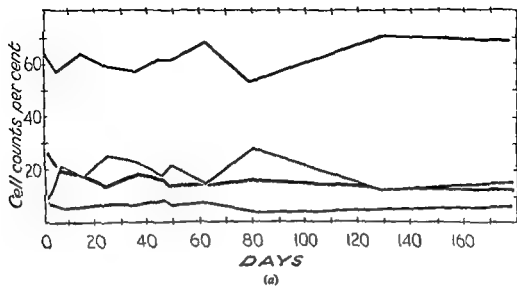
It is important to note that the presence of differentiated cells, whether initially characteristic of the tumour or induced by irradiation, is to be regarded as a favourable sign from the point of view of radio-curability. On the other hand, a mere reduction in viable cells, without such evidence of change in type, is not encouraging; the growth is certainly radio-sensitive but is not curable by irradiation.

In many of the cases studied by these authors clinical healing was obtained in spite of an unfavourable histological response. In such cases, however, early recurrence took place. It is to be remembered that practically all tumours shrink to some extent under treatment—possibly as an indirect result of irradiation in reducing the vascular supply—but such clinical improvement is of no significance if viable tumour cells remain at the site. The agreement between histological and clinical findings increases with the lapse of time; in 150 cases examined by Glücksmann and Spear although this was present in only 50 per cent 4 months after treatment, the figure rose to 80 per cent after 2 years.

An important aspect of this work is that it opens up the possibility of early assessment of the effect of irradiation. The changes described may be recognized within 3 weeks from the commencement of treatment, and with further experience it may be possible to decide by means of a test irradiation whether surgical excision or irradiation therapy gives the greater promise of success.

*Effects of
irradiation*

*Agreement of
histological
and clinical
findings*



Carcinoma of the cervix (a) Chart of an unsuccessfully treated case after tr
mitosis
treated
mitotic
of degenerating cells (14 biopsies) (*Brit J Radiol*)

PLATE II



REFERENCES

- Broders, A. C. (1920) *J. Amer. med. Ass.*, **74**, 656.
 — (1932). *N. Y. St. J. Med.*, **32**, 669.
- Butlin, H. T. (1909) *Brit. med. J.*, **1**, 1.
- Dukes, C. (1946). *Rep. Brit. Emp. Cancer Campaign*, p. 58. (Unpublished.)
- Ewing, J. (1940). *Neoplastic Diseases. A Treatise on Tumors*, 4th ed. Philadelphia; Saunders.
- Glucksmann, A. (1941). *Brit. J. Radiol.*, **14**, 187.
 — (1946) *Brit. med. Bull.*, **4**, 26.
 — and Spear, F. G. (1945). *Brit. J. Radiol.*, **18**, 313.
- Handley, W. S. (1922) *Cancer of the Breast*, 2nd ed. London; Murray.
- McFarland, J. (1942). *Amer. J. med. Sci.*, **203**, 502.
- Patey, D. H., and Scarff, R. W. (1928). *Lancet*, **1**, 801.
- Turner, G. G. (1937). *Proc. R. Soc. Med.*, **30**, 301.
 — (1939). *Trans. St. John's Hosp. Derm. Soc. Lond.*, **28**, 93.
- Willis, R. A. (1934). *The Spread of Tumours in the Human Body*. London; Churchill.
- [References to other titles are given under Neoplasms—Innocent and Malignant, in the Index Volume]

NERVES—CRANIAL

BY JOHN E. A. O'CONNELL, M.S., F.R.C.S.
NEURO-SURGEON TO ST. BARTHOLOMEW'S HOSPITAL, LONDON

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1. INTRODUCTION

241.] The cranial nerves form part of the peripheral nervous system and, with the exception of the optic nerve, are structurally the same as other peripheral nerves. However, their topography invests them with a particular importance. Close relationship to the skull leads to their frequent involvement in fractures and other lesions of the skull base. Cranial nerve injury therefore forms an important aspect of head injuries in general. Again, the origin and course of these nerves within the skull leads to their involvement in intracranial disease, and evidence of this may give diagnostic aid of a localizing nature. Certain of the cranial nerves are subject to recurring paroxysmal disturbances of function not seen in other peripheral nerves, leading to the production of severe symptoms—trigeminal neuralgia, glossopharyngeal neuralgia and Ménière's disease. Finally, in the optic nerve there is exposed for ophthalmoscopic inspection a portion of the central nervous system in which changes may be produced not only by local disease but also by distant intracranial lesions, and in which intracranial hypertension may quickly reveal itself by the development of papilloedema.

2. TRAUMATIC LESIONS OF THE CRANIAL NERVES

Of the cases of head injury which require admission to hospital about 15 per cent show evidence of a cranial nerve lesion (Turner, 1943). This is due in part to the close anatomical relationship of the nerves to the skull as they

pass through their cranial foramina and to the fact that basal fractures of the skull frequently involve one or more of these foramina. However, it is noteworthy that a cranial nerve lesion may complicate a head injury in the absence of a fracture. *Basal fractures*

(1) Olfactory nerve

Injuries of the olfactory nerve account for about half the post-traumatic cranial nerve palsies. The lesion results from two types of violence.

(a) A fracture involving the anterior fossa which passes through the cribriform plates and is associated with contusion or laceration of one or both of the olfactory bulbs. *Anterior fossa*

(b) Occipital injuries, which may produce a fracture radiating into the foramen magnum but do not produce bony injury anteriorly, may also be complicated by anosmia (Leigh, 1943). It is suggested that in these cases olfactory nerve filaments are torn by the shearing stresses applied to them when the cerebrum is displaced within the skull. *Anosmia*

The result of such injuries will be a partial loss (hyposmia), or complete loss (anosmia), of the sense of smell. Sometimes olfactory hallucinations are present (parosmia). The sense of taste is frequently, but not always, affected owing to the important part played by olfaction in the appreciation of flavour. The prognosis in cases of involvement of the olfactory nerve after head injuries is bad. The presence of anosmia in a patient with post-concussional symptoms will be helpful when attempting to assess the severity of the injury. *Bad prognosis*

(2) Facial nerve

The facial nerve was involved in 20 per cent of Turner's series (1944). Almost invariably there is an associated fracture of the temporal bone with injury either to the ear or to the eighth cranial nerve or to both. In approximately half of the number of these cases the facial paralysis is of immediate onset, and in the others is delayed for a varying period after the injury. The immediate paralysis results from contusion or laceration of the nerve in the fracture line, while the delayed form probably results from oedema of the nerve within its confined bony canal. The prognosis in both types is good, being slightly better in delayed lesions, in which presumably the connective tissue framework of the nerve always remains undisturbed. *Good prognosis*

Treatment

Treatment consists in splinting the lower face, in order that the unopposed orbicularis oris of the opposite side may not produce severe deformity and stretching of the paralysed muscle fibres, and massage and galvanic stimulation for these muscles is of benefit. In those cases in which spontaneous recovery does not occur, and in which the lesion is accessible, a nerve graft to replace the injured segment may give a good result—it is one of the most successful nerve grafts. (See Facial Palsy in Vol 4, p.1.) If the graft is not possible a facio-hypoglossal anastomosis may be carried out with good prospect of beneficial results. *Nerve graft*

(3) Third, fourth and sixth nerves

The nerves to the ocular muscles, the oculomotor, trochlear and abducens nerves are involved almost as frequently as the facial nerve, and fractures of the related portion of the skull base are a frequent accompaniment. The

Diplopia

prognosis for functional recovery is good. It must be borne in mind that diplopia following head injuries may be the result of direct injury to the ocular muscles or even to displacement of the globe occasioned by injury to the bony wall of the orbit.

Hutchinson's pupil

A very important variety of post-traumatic oculomotor palsy is that which gives rise to Hutchinson's pupil. The outstanding feature here is the fixed dilatation of one pupil. In patients exhibiting this sign and in whom prompt treatment of the causative lesion is instituted with subsequent recovery, it will frequently be seen later that there is a complete lesion of the corresponding third nerve so that not only are the light and convergence reactions of the

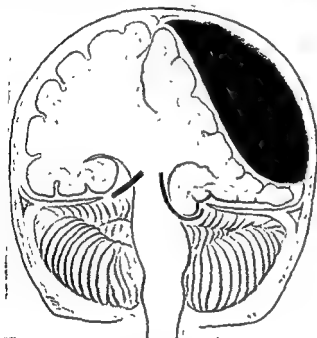
*Lesion of third nerve**Herniation of hippocampal gyrus*

FIG. 108.—Diagram to show how herniation of the hippocampal gyrus stretches the third cranial nerve, causing Hutchinson's pupil.

pupil absent, but there is an external strabismus and paralysis of the extra-ocular muscles supplied by the third nerve. The cause of the lesion, as has been demonstrated experimentally by Reid and Cone (1939), is a herniation of the hippocampal gyrus of the temporal lobe through the tentorial opening with stretching of the oculomotor nerve beneath it. Such a tentorial pressure cone will sometimes be bilateral, but it will develop earlier and be larger on the side of the lesion, be this a post-traumatic haemorrhage or an intracranial tumour.

This is the explanation of

the valuable dictum that in post-traumatic haemorrhage without any other localizing signs the dilated pupil is on the same side as the lesion (Fig. 108).

(4) Optic nerve

In 10 per cent of a large series of post-traumatic cranial nerve lesions the optic nerve was involved. Here the trauma has commonly been applied to the frontal or anterior temporal region on the side of the injured nerve. Occasionally there is an associated fracture involving the optic foramen, but usually this is lacking. In such cases it may be that the nerve as it passes through the optic foramen is injured by a transient deformation of the bony ring which does not lead to fracture. It has also been suggested that in these cases the primary injury may involve the vascular supply of the nerve and that the visual disturbance is secondary to this rather than to a direct nerve injury. The prognosis is for the most part bad—though in the less severe injuries improvement in vision may occur over a period of several months.

Prognosis

3. DIAGNOSTIC SIGNIFICANCE OF CRANIAL NERVE LESIONS

While evidence of disturbed function of a particular cranial nerve will always have an intrinsic importance in view of the disability which it occasions, the disturbance may possess considerable secondary importance. This is particularly so when an anatomically related group of nerves is involved, since the signs may point clearly not only to the localization of the lesion but to its pathological nature.

In the first place, since the cranial nerves will reach the exterior of the skull by piercing its base, it is clear that they will be involved especially in basal disease processes. When it is recalled that the foramina of the exit of these cranial nerves occupy but a relatively small central area of the skull base (Fig. 109), *Relative to skull base*

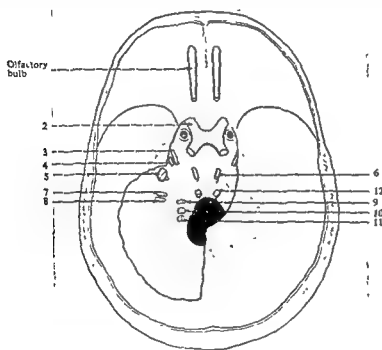


FIG. 109 — The cranial nerves in relation to the skull base

the value of such localizing evidence becomes clear, and it can be readily understood why diffuse processes, whether traumatic, inflammatory or neoplastic, may produce multiple cranial nerve lesions.

It so happens that in the case of more localized pathological processes within the skull there is a close relationship between the site of the lesion and its pathology. In such cases the cranial nerve lesions will therefore indicate not only the site of the lesion but will also suggest its nature.

(1) Intracranial aneurysm

Thus a basal aneurysm arising from the supraclinoid portion of the internal carotid artery or the circle of Willis or one of its main branches will be particularly related to the oculomotor nerve and the optic chiasma. The occurrence of an oculomotor palsy with or without an associated field defect, *Oculomotor palsy*

together with a history of recurring headaches, and possibly attacks of unconsciousness with subsequent evidence of meningeal irritation, will strongly suggest the presence of such a basal aneurysm.

(2) Basal neoplasms

The various neoplasms which grow at the skull base have particular sites of origin, and each therefore tends to affect a different group of cranial nerves. Thus unilateral anosmia with ipsilateral optic atrophy points to a meningioma arising from the olfactory groove; the association of trigeminal neuralgia with diminished sensibility in the territory of this nerve may indicate a meningioma arising in Meckel's cave; or the progressive involvement of the eighth cranial nerve together with the fifth, seventh, ninth, tenth and eleventh cranial nerves and added evidence of a cerebellar lesion on the same side with intracranial hypertension, point unmistakably to the diagnosis of an acoustic neurinoma.

(3) Nasopharyngeal tumours

It must be remembered that certain tumours of the nasopharynx may extend deeply to and through the skull base while still of such small superficial extent as to be symptomless at their site of origin. Various cranial nerve palsies may thus be produced. In anteriorly placed tumours the triad of Jacod may arise—amaurosis, ophthalmoplegia and trigeminal neuralgia. In posterior invasive growths other syndromes may arise—for example, that of Trotter in which palatal paralysis, neuralgia of the mandibular division of the fifth nerve and deafness from Eustachian obstruction are combined. This third group of cases indicates the importance of nasopharyngeal examination in patients with isolated or grouped cranial nerve palsies. In these patients skiagrams of the skull may show the erosion of its base, and the projection of a mass into the nasopharyngeal air shadow will at times be visible.

The importance of cranial nerve lesions as an indication of the site and pathology of intracranial disease is therefore clear. It must be remembered, however, that at times, in the case of intracranial neoplasms, such evidence of the localization of a lesion may be misleading as the localizing sign is a false one. This is particularly so when evidence of raised intracranial pressure has preceded the development of the nerve palsy by a considerable interval. Paralysis of the abducens is the commonest of such false localizing signs, and is to be regarded merely as evidence of raised intracranial pressure alone, since the tumour causing the hypertension in such a case may be anywhere within the skull, and is often far removed from the nerve concerned. The explanation of the frequency of paralysis or paresis of the sixth nerves at one time favoured was that the long intracranial course and exposed position of these nerves

at the base of the skull suggested that a backward shift of the brain-stem occasioned by intracranial tumours would cause stretching of the longitudinally disposed abducens and interference with their functions. Subsequently Cushing (1910-11) produced evidence that in many cases this most common of false localizing signs is due to strangulation of the abducens by transverse branches of the basilar artery. The vessels generally overlie the nerves and may cut deep grooves in them as well as in the brain-stem when the latter is enlarged or deformed by the

*Unilateral
anosmia*

Meningioma

*Triad of
Jacod*

*Nasopharyn-
geal
examination*

*Intracranial
neoplasms*

*Paralysis
of abducens*

*Enlarged
brain-stem*

presence of an intracranial neoplasm, especially one in the posterior cranial fossa. The third, fifth and seventh cranial nerves also may exhibit disturbances of conduction which are in the nature of false localizing signs.

4. NEURALGIA INVOLVING THE FACE AND MOUTH

Certain of the cranial nerves are subject to recurrent paroxysmal disturbances of function—largely, though not necessarily, of a subjective kind—for which no adequate explanation has been suggested. Trigeminal neuralgia is the most frequent of these, but a very similar neuralgia may affect the glossopharyngeal nerve, and it is thought that otitic pain may occasionally be the result of neuralgia of the pars intermedia of the facial nerve. Clonic facial spasm and the severe paroxysmal vertigo of Ménière's disease are in some respects similar to these neuralgias, though in the case of Ménière's disease there is objective evidence of aural abnormality in the progressive deafness and constant tinnitus which are associated with the chief symptoms. Certain of these conditions of obscure aetiology (excluding trigeminal neuralgia which is described on p. 251) will now be considered individually.

Tinnitus

(1) Glossopharyngeal neuralgia

This condition is characterized by paroxysmal attacks of severe lancinating pain in the sensory distribution of the glossopharyngeal nerve (the back of the tongue, the lateral pharyngeal wall and the ear). As in the case of trigeminal neuralgia the pain is frequently produced by the stimulation of trigger points—especially on the pharyngeal wall during the act of deglutition. In an attack of pain the patient will endeavour to avoid eating and drinking and will even allow saliva to trickle from his mouth in an effort to avoid stimulation of the trigger point.

The one condition with which glossopharyngeal neuralgia may be confused is trigeminal neuralgia. The distribution of pain and the position of the trigger points should make differentiation simple. In glossopharyngeal neuralgia pain is produced by swallowing; in trigeminal neuralgia it is caused by chewing. In case of doubt the effect of anaesthetizing the mucosa of the throat with cocaine should indicate the diagnosis.

Symptoms

Operative treatment

In the treatment of glossopharyngeal neuralgia some have advised that the nerve be sectioned in the neck—or even by an approach through the floor of the tonsillar fossa. Such operations correspond to attacks upon the peripheral branches of the trigeminal nerve in tic douloureux and are no more likely to lead to permanent relief. The ideal operation is intracranial section of the glossopharyngeal nerve immediately proximal to the jugular foramen. Dandy showed that the same approach may be used for intracranial section of the fifth, eighth and ninth cranial nerves. With slight modification it may be utilized in the treatment of the intractable pain of unilateral malignant disease of the mouth by sectioning the upper cervical posterior roots in addition to the fifth and ninth nerves.

Ideal operation

Detailed technique.—A flap of soft tissues is turned downward from between the mastoid process and the external occipital protuberance and a small sub-occipital craniotomy is made—the bone removal being carried superiorly

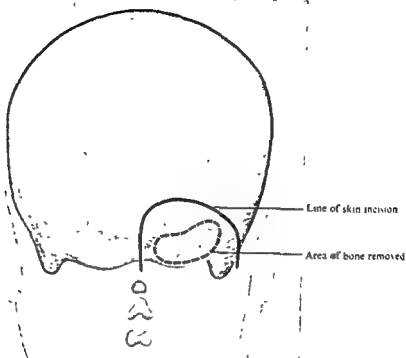


FIG. 110.—Position of incision and area of bone removal in the exposure of the eighth and ninth cranial nerves in the posterior cranial fossa

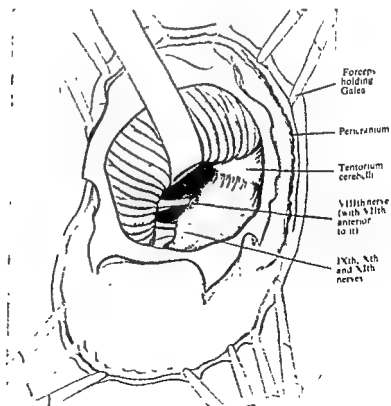


FIG. 111.—Operative exposure of the eighth and ninth cranial nerves completed.

and laterally to expose the lateral sinus but if possible avoiding injury to the mastoid air cells (Fig. 110). The dura is incised and the cisterna magna is evacuated to reduce tension. The cerebellar hemisphere, protected by moist lintine, is retracted medially to expose the posterior surface of the petrous temporal bone with the tentorium cerebelli attached to its superior margin. The internal auditory meatus is then seen—the eighth nerve here lying posterior to the seventh. Inferiorly, the ninth, tenth and eleventh cranial nerves are seen converging on the jugular foramen (Fig. 111). The ninth cranial nerve is the smallest of these and is superiorly placed, having a distinct dural opening above that which gives passage to the other two nerves. The nerve is picked up and sectioned with a right-angled knife. Complete haemostasis is ensured, the dura is closed, and then the superficial tissues are closed in layers, muscle, galea and skin.

(2) Geniculate neuralgia and facial spasm

The occurrence of pain in the ear and its immediate neighbourhood in association with geniculate herpes has led to the suggestion that paroxysms of pain in this region, in the absence of herpes, may be examples of geniculate neuralgia. The condition is one of great rarity and care must be taken to exclude the possibility of the pain being due to local disease or referred from the pharynx. Cases are on record in which section of the seventh nerve has relieved such pain.

Relief of pain

The motor territory of the seventh nerve may at times be the seat of recurring paroxysmal disturbance of function with the occurrence of clonic facial spasm. This has sometimes been associated with facial pain of either geniculate or trigeminal type. The condition is characterized by involuntary contractions of some portion of the facial musculature which spread until the whole of one side of the face is involved in a spasmodic contracture which can neither be controlled nor imitated by the patient. It is thus in no way related to habit spasm and neurosis, and interruption of the facial nerve may be required for its cure. This may be produced by alcohol injection, stretching of the nerve, or section which may be combined with anastomosis of the peripheral divided end of the seventh nerve to the central end of either the hypoglossal or the spinal accessory nerve.

Clonic facial spasm

Cure

(3) Ménière's disease

There remains for discussion another paroxysmal disturbance of cranial nerve function, recurrent aural vertigo or Ménière's disease. Here, too, the aetiology is uncertain though the association with giddiness of deafness and tinnitus in the affected ear does point to a lesion involving the eighth cranial nerve or its end organs. Various suggestions as to the nature of this lesion have been made. The frequently quoted statement that Ménière had found a haemorrhagic exudate in the semicircular canals in these cases is erroneous. Focal infection, allergy and a disturbance of water metabolism leading to gross dilatation of the endolymphatic system are among the hypotheses upon which therapy has been based. Dandy (1941) stated that in over 20 per cent of the cases which he treated by intracranial section of the vestibular nerve there was a local lesion, either a neoplasm or an abnormal vessel on the course of the nerve, which he considered to be the cause of the symptoms.

Nature of lesion

(a) Clinical features

Clinically the patient, somewhat more often a man than a woman, frequently in middle life, notices a progressively increasing unilateral deafness with a constant, though fluctuating, tinnitus in the same ear. After a varying period recurrent attacks of severe vertigo appear. The onset of such attacks may be so sudden and severe as to cause the patient to fall, though at times the effect is less dramatic. During the attack there is a feeling that either he or his surroundings are rotating, and as this is aggravated by movement, he will try to remain immobile with closed eyes. Vomiting is frequently associated with the attacks and the patient may be prostrated for hours. In the intervals between paroxysms slight unsteadiness of gait and stance may be present. Examination reveals only diminished auditory acuity in the affected ear with sometimes a reduced labyrinthine response in addition.

(b) Diagnosis

In diagnosis the numerous causes of vertigo must be considered. The symptom may be due to causes in the external, middle or internal ear, the eighth cranial nerve, brain-stem, cerebellum, and indeed at higher levels in the nervous system, but a careful history and examination will frequently determine the site of the lesion. In the case of lesions of the eighth nerve itself it may be impossible to decide whether there is a small acoustic neuroma arising from the nerve or whether the case is one of Ménière's disease. This is an argument in favour of intracranial section of the vestibular nerve in the treatment of the latter condition rather than destruction of the labyrinth, which would not reveal the lesion and would lead to delay in its correct diagnosis and treatment.

(c) Treatment

Surgical treatment will be indicated only when conservative measures fail. These include the administration of phenobarbitone, limitation of the intake of fluid, and the use of ammonium chloride instead of sodium chloride to season food. When surgery is indicated it may take the form of a destruction of the labyrinth or intracranial section of the vestibular portion of the eighth nerve. The latter procedure has the advantage of allowing exposure and removal of such a causative lesion as a tumour, and of sparing such hearing as is retained in the affected ear—particularly important should the opposite ear be deaf. It should therefore be preferred if the possibility of a neoplasm exists or when there is fair preservation of hearing in the affected ear, the opposite one being deaf. The operation is performed through an approach similar to that described for the ninth nerve in the posterior fossa (see p. 213). The eighth nerve lies posterior to the seventh and must be elevated from the former before cutting across its superior (or cephalad) two-thirds, which contain the vestibular fibres. (See Ear—Internal Ear, Chronic Infection (Non-suppurative), Vol. 3, p. 296.)

5. TUMOURS OF THE CRANIAL NERVES

The subject of the tumours of the cranial nerves is but a part of the larger one of intracranial tumours in general—though, of course, at times a neoplasm arises from the extracranial portion of one of these nerves. While the intracranial portion of any of the nerves may be involved, two alone merit mention here—the second and the eighth nerves.

(1) Second nerve

As the second nerve is in reality an outgrowth of the brain it is only to be expected that a tumour arising from it should be gliomatous, and frequently it is a spongioblastoma polare. Both the orbital and intradural portions of the nerve may be involved and there is a marked tendency for spread (often microscopical) of the growth to occur in the long axis of the nerve with chiasmal involvement. An early transfrontal approach is indicated with section of the nerve as far posteriorly as possible. By division of the nerve behind the globe the eye may be spared in some cases.

Gliomatous
tumour**(2) Eighth nerve**

Neoplasms of the eighth nerve arise from the vestibular portion of the complex and are, like tumours of other peripheral nerves, neurofibromas or neurinomas thought to originate in the neurilemmal sheath. There is usually a single tumour, but bilateral auditory nerve tumours may be found especially in association with the manifestations of von Recklinghausen's disease. Solitary auditory nerve tumours are of common occurrence and make up some 10 per cent of most large series of intracranial neoplasms. Cushing (1917) called attention to their characteristic clinical picture which depends, as has already been indicated, upon the origin of the tumour from the eighth nerve, upon its proximity to other cranial nerves, and to the cerebellar peduncles, and upon the obstruction of the bottle-neck in the cerebrospinal fluid pathways at the tentorial opening. In the early years of neurosurgery, because of the very high mortality which was associated with attempts to remove these growths completely, Cushing believed that no more than an intracapsular removal was indicated. Dandy (1925), however, had for long recommended total excision because of the almost inevitable recurrence after a more conservative operation, and now an increasing number of neurosurgeons are in agreement with him. With modern advances in technique, in particular by operating with the patient in the sitting position, total excision of the growth is being done with increasing frequency, and in certain cases preservation of the facial nerve may be possible. However, if the nerve is sacrificed, a subsequent hypoglossofacial anastomosis can be performed and will frequently be followed by a most satisfactory return of tone and mobility in the facial muscles.

Auditory
nerve
tumoursTotal
excision
of growth

(See also article on Brain—Tumours and Technique, Vol 2, p 420)

REFERENCES

Collins, J. (1933) *Proc. Roy. Soc. Med.*, 26, 100.

.. . .

.. . .

.. . .

* the Syndrome of the Cere-

bellopontile Angle. Philadelphia and London; Saunders.

Dandy, W. E. (1925) *Surg. Gynec. Obstet.*, 41, 129.

— (1941). *Ibid.*, 72, 421.

Leigh, A. D. (1943) *Lancet*, 1, 38.

Reid, W. L., and Cone, W. V. (1939) *J. Amer. med. Ass.*, 112, 2,030.

Turner, J. W. A. (1943). *Brain*, 66, 140.

— (1944) *Lancet*, 1, 756.

[References to other titles are given under Nerves—Cranial, in the Index Volume. The subject is also dealt with under the heading of Cranial Nerve Affections in the *British Encyclopaedia of Medical Practice* (1937), Vol 3, p. 470.]

NERVES, PERIPHERAL—INJURIES

BY ATHOL PARKES, M.B., F.R.C.S.ED.
SURGEON-IN-CHARGE, PERIPHERAL NERVE INJURY UNIT, KILLEARN
HOSPITAL, GLASGOW; ASSISTANT ORTHOPAEDIC SURGEON, WESTERN
INFIRMARY, GLASGOW

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1. DEFINITION

242.] The peripheral nerve injuries dealt with in this section are those of the main nerve trunks of the limbs. Causalgia as a complication of nerve injury, injuries of the brachial plexus, and the conditions attributed to pressure on the nerves at the thoracic outlet, such as the costo-clavicular syndrome and cervical rib, are dealt with elsewhere.

2 AETIOLOGY

Although the majority of peripheral nerve injuries are due to direct trauma to the nerve trunks by external violence or by fractures of the long bones, more indirect forms of nerve injury such as traumatic ulnar neuritis and damage caused by ischaemia will be considered. Medical conditions such as peripheral neuritis do not come within the scope of this section.

3. ANATOMY AND PHYSIOLOGY

It is assumed that the surgeon has a sound knowledge of the gross anatomy of the main nerve trunks in the limbs but, as the diagnosis and treatment of injuries to the peripheral nerves depend very largely on such knowledge, some space will be devoted to anatomical features under "Clinical Picture" and "Operative Technique". It may be well, however, to enumerate the various types of fibres present in the peripheral nerves and to indicate the func- *Types of fibres* tions of each, because the clinical picture presented by any case of complete division of a nerve has features indicating loss of conductivity in all fibres, whereas in certain other lesions some fibres tend to be more vulnerable than others.

TYPES OF FIBRES	EFFECTS OF LOSS OF CONDUCTIVITY
<i>Efferent</i>	
(1) Motor fibres to skeletal muscles	Paralysis, wasting and certain changes in the electrical reactions of the muscles supplied
(2) Autonomic fibres	
(a) Vasomotor	Vasodilatation and increased heat, followed, after some weeks, by vasoconstriction and coldness of the denervated area
(b) Sudomotor	Loss of sweating in the area of distribution
(c) Pilomotor	Loss of "goose-skin" reaction to cold

Afferent

- | | |
|---|---|
| (1) Sensory fibres from the skin | Loss of the various forms of cutaneous sensation. The area of loss to light touch is usually greater than that to pain because in the former there is less overlap from adjacent intact areas |
| (a) Light touch | |
| (b) "Fast" pain | |
| (c) "Slow" pain | |
| (d) Temperature | |
| (2) Sensory fibres from deep structures | Loss of deep pressure, muscle and bone sensation |
| (3) Proprioceptive fibres | Loss of postural sensibility |

Wasting of the digital pulps, increased curvature and brittleness of the nails, change in joint structure producing stiffness, and liability to ulceration of the skin after trivial injuries have all been attributed to the loss of efferent trophic nerve fibres. These alterations in tissue nutrition are to be regarded as the result of disuse, sensory loss and vascular changes.

*Trophic
function of
nerve fibres*

4. MORBID ANATOMY

The following table gives a reasonably satisfactory classification of nerve lesions, and covers all types likely to be encountered as a result of injury.

Types of nerve lesions

- (1) Anatomical division
 - (a) With destruction of essential structures—neurotmesis (Seddon, 1943)
 - (b) Complete
 - (c) Partial
- (2) Compression lesions
 - (a) Lesion in continuity—axonotmesis (Seddon, 1943)
 - (b) Transient physiological block—neurapraxia (Seddon, 1943)
- (3) Traction lesions
- (4) Ischaemic lesions
 - (a) Caused by traumatic arterial spasm
 - (b) Caused by pressure beneath the deep fascia
- (5) Chemical lesions

Mixed lesions

In making the above classification, however, it must be clearly understood that, in any particular case, more than one type of nerve lesion may exist; for example, not only may one trunk have suffered anatomical division and the conductivity of another have been abolished because of a compression lesion, but more than one type of lesion may be present in an injury to a single trunk. Consequently, although it may be possible to diagnose the type of nerve lesion with some confidence purely on clinical grounds, it is often necessary to carry out an exploratory operation as part of the diagnostic procedure, and even then it may not be possible to be certain as to which type of lesion is the predominating one.

*Diagnostic
exploration*

(1) Anatomical division—neurotmesis

This type of lesion usually occurs as a result of incised, lacerated or stab wounds, though it may be found in closed injuries, for example, as a

complication of a fracture. There is, of course, peripheral degeneration of the nerve fibres distal to the point of division, and complete loss of conductivity in all types of fibres, unless the division involves only part of the nerve trunk.

(2) Compression lesions

The epineural sheath remains intact in these cases and damage to the contained nerve fibres varies in degree according to the severity of the injury. The essential structures may be completely destroyed and replaced by fibrous tissue so that, in effect, the condition is one of anatomical division (neurotmesis). In less severe injuries the internal architecture of the nerve may be preserved but the axons so badly damaged that peripheral degeneration takes place (axonotmesis); or the damage may be so slight that it causes only a transient physiological block (neurapraxia). In neurotmesis recovery can occur only after resection and suture. In axonotmesis recovery is spontaneous but, as it depends on regeneration, is slow and often incomplete. In neurapraxia conductivity is generally completely restored within 5 or 6 weeks. In partial compression lesions, motor fibres tend to be more vulnerable than the sensory fibres. The common traumatic ulnar neuritis at the elbow is a chronic form of compression lesion.

*Degrees of
compression
lesions*

Recovery

(3) Traction lesions

Occurring most commonly in the brachial plexus and in the lateral popliteal nerve, these lesions affect a considerable length of the nerve trunks. They can be likened to the state of affairs in an overstrained rope, in which different strands give way at various places along the length of the strained portion. There is much intraneural scarring, and motor fibres tend to be affected more than sensory fibres. Peripheral degeneration of the affected fibres often takes place and spontaneous recovery, if it does occur, is slow and usually incomplete.

(4) Ischaemic lesions

Ischaemia of the nerve trunks sufficient to produce not only loss of conductivity but, if prolonged, peripheral degeneration can occur either as a result of a local traumatic arterial spasm—with or without Volkmann's ischaemic necrosis of adjacent muscles—or when the nerve trunks lie within the fascial envelope of a tensely swollen limb (Parkes, 1945). Although in such cases the affected nerve trunks may be situated in the proximal part of the limb, the effects of the lesion tend to be confined to the more distal parts and are typified by a "glove" or "stocking" type of anaesthesia, and paralysis of the intrinsic muscles of the hand or foot. Sensory fibres tend to be more susceptible to ischaemia than motor fibres, but the "slow" (or slow-conducting) pain fibres often survive, causing a delayed pain response to pin-prick and pressure. Spontaneous recovery is slow and often incomplete.

*Site of
lesion and
of effects*

*Relative
susceptibility
of fibres*

(5) Chemical lesions

Such lesions are due to accidental injection into a nerve trunk of certain chemical substances used therapeutically. There is much intraneural scarring together with peripheral degeneration of fibres. Recovery, as a rule, is slow and incomplete.

5. CLINICAL PICTURE

(1) Clinical examination

The necessity for carrying out a brief examination to exclude the presence of a nerve lesion in any case of more than trivial injury to a limb, need hardly be stressed. Usually a few simple tests of the motor and sensory functions of the main nerve trunks are all that is required, but when these reveal that there has been some interference with conduction an attempt should be made to assess the type of injury likely to be present, for this will affect the treatment to be adopted and the prognosis. As a general rule, in the presence of signs of complete interruption of conduction in a nerve trunk in the vicinity of an open wound, it should be assumed that the nerve has been completely divided, until there is evidence to the contrary. In closed injuries, on the other hand, it is justifiable to assume that the lesion is probably one of the other types with an intact nerve sheath. The method of injury may give a clue as to the type of lesion likely to be present—for example, a traction lesion—and signs of damage to other structures, as by ischaemia, may also provide valuable evidence. Palpation of the nerve trunks will frequently reveal the site of the lesion, for in thin subjects considerable lengths of the main nerve trunks can easily be felt through the skin, especially when they are rendered taut owing to scarring at the site of injury. In addition to the above points, the nature of any deformity which may be present, as well as the condition of the joints, muscles and skin, should be assessed before testing the specific functions of the suspected nerve. As regards the latter, sufficient information can generally be obtained by testing for voluntary power in all the muscles supplied by the nerve and for sensory loss in its cutaneous distribution. Muscle power is recorded, preferably on a chart, and the activity of each muscle should be indicated by a figure from 0 (complete paralysis) to 5 (full power). Sensory symptoms, such as loss of response to light touch and pin-prick, should also be noted.

(2) Individual nerve lesions

The clinical picture presented by any case of injury to a particular nerve naturally depends on the type of lesion and the level of the injury. The following descriptions refer mainly to complete lesions. Considerable variations occur when the lesion is incomplete, and the picture may be modified, too, by anatomical variations in the distribution of the individual nerves. Complete lesions are frequently masked by the ability of some patients to develop trick

(a) *Upper limb* (see also Brachial Plexus in vol. 2, p. 335).

(i) *Circumflex nerve*.—The motor and sensory distribution is indicated in Figs. 112 and 113. There is no obvious deformity unless wasting of the deltoid muscle has taken place. Paralysis of the deltoid results in impaired abduction of the shoulder, but some patients have a surprisingly good power of abduction by the supraspinatus and pectoralis major muscles, and it is necessary to

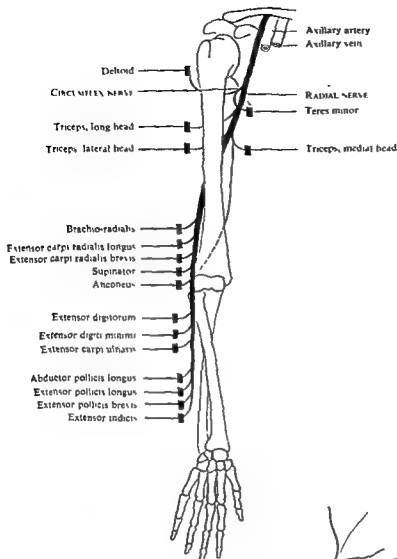
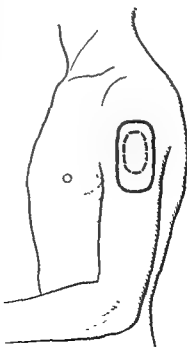


FIG. 112—Diagram of the circumflex and radial nerves and the muscles they supply.

FIG. 113—The approximate area within which sensory changes may be found in lesions of the circumflex nerve. Light touch: heavy line. Pin prick: broken line.



Differential diagnosis

assess the degree of activity in the deltoid muscle itself. Sensory loss is restricted to a small area over the deltoid (Fig. 113). The spinati and brachioradialis should be tested to exclude a lesion of the upper root (C.5) of the plexus.



FIG. 114.—The approximate area within which sensory changes may be found in high lesions of the radial nerve. In most cases there is only a small area of anaesthesia on the dorsum of the hand.

(ii) *Radial nerve* (including posterior interosseous).—The motor and sensory distribution is indicated in Figs. 112 and 114. The radial nerve is predominantly motor, and in lesions below the level of the radial groove of the humerus there may be very little sensory loss. Wrist-drop is the obvious deformity. The patient is unable to dorsiflex the wrist or to extend the fingers at the metacarpo-phalangeal joints, but active extension of the interphalangeal joints is retained (ulnar nerve). There is loss of active extension and of abduction of the

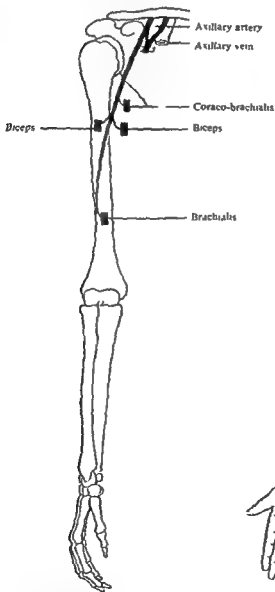
Motor loss

FIG. 115.—Diagram of the musculo-cutaneous nerve and the muscles which it supplies

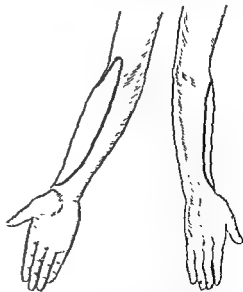


FIG. 116.—The approximate area within which sensory changes may be found in lesions of the musculo-cutaneous nerve

thumb, though some active extension of the interphalangeal joint may be carried out by the abductor pollicis brevis (median nerve). Even in high lesions the long head of the triceps is seldom affected. Lesions of the posterior cord of the plexus (deltoid paralysed) and wrist-drop from other causes, such as lead poisoning, must be excluded. *Differential diagnosis*

(iii) *Musculo-cutaneous nerve*.—The motor and sensory distribution is indicated in Figs. 115 and 116. There is no obvious deformity unless wasting of biceps has occurred. Paralysis of the biceps and brachialis muscles generally abolishes the power of active flexion of the elbow against gravity, but weak flexion can still be carried out by brachioradialis and that part of the brachialis muscle supplied by the radial nerve. There is subjective numbness along the radial border of the forearm (Fig. 116) but the objective loss is often slight. *Motor and sensory loss*
A lesion of the upper trunk of the plexus, in which deltoid, spinati and

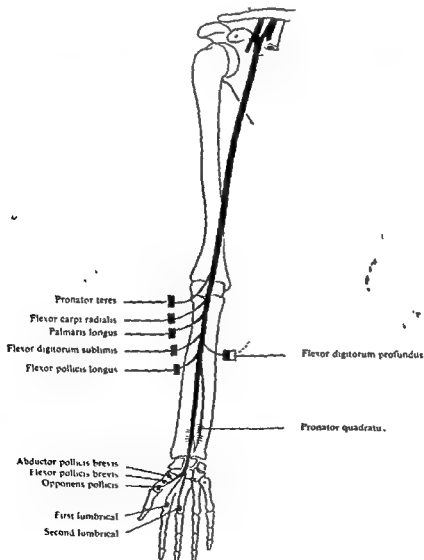


FIG 117.—Diagram of the median nerve and the muscles which it supplies.
B.S.P. 6—15

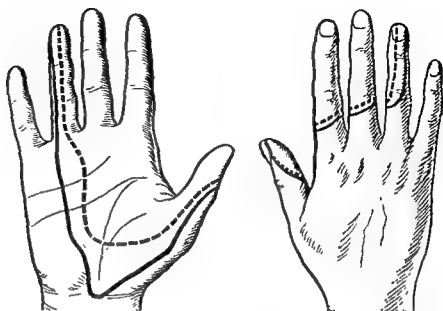


FIG 118 —The approximate area within which sensory changes may be found in lesions of the median nerve. The area of sensory loss is usually smaller than this, and especially in lesions at the wrist the area of loss to pin prick may be confined to the distal halves of the radial three digits. Light touch: heavy line. Pin prick: broken line.



brachioradialis are affected, or of the lateral cord of the plexus, in which pronator teres and flexor carpi radialis are affected, must be excluded. *Differential diagnosis*

(iv) *Median nerve.*—The median nerve, arising by two heads from the plexus, supplies the muscles indicated in Fig. 117. The sensory distribution (Fig. 118) involves what is probably the most important area for cutaneous sensation in the whole body. Deformity is inconspicuous in a case of recent injury (Fig. 119), but in an old-standing case the combination of the ape-like position of the thumb with wasting of the thenar eminence (simian hand), together with the extended position and wasting of the digital pulp of the index finger (pointing index) is typical. In lesions above the elbow the motor paralysis causes marked weakness of pronation of the forearm and inability to flex the interphalangeal joints of the thumb and index. Active opposition of the thumb is usually weak or absent. It should be stressed that active palmar flexion of the wrist can still be carried out by flexor carpi ulnaris and abductor pollicis longus, and there is good active flexion of the inner three fingers (flexor digitorum profundus muscle). Weak flexion of the index finger may accompany flexion of the other three fingers due to spread from the ulnar portion of flexor profundus. The loss of power in the thenar muscles depends largely on the extent of their supply from the ulnar nerve, and only abductor pollicis brevis may be affected (Higbet, 1942). The sensory loss also varies considerably but, even when small in extent, it is extremely disabling. *Importance of sensory area*
Motor loss
Anatomical variations

Traumatic median neuritis (Zachary, 1945) is a chronic compression lesion of the median nerve at the wrist. It is due to pressure on the nerve by the proximal border of the flexor retinaculum. The main clinical features are wasting and weakness of abductor pollicis brevis and sensory changes in the distribution of the median nerve. The condition can be relieved, or at least prevented from getting worse, by division of the constricting proximal border of the flexor retinaculum. *Clinical features*
Treatment

Causalgia, although not by any means a common condition, occurs relatively frequently as a complication of partial lesions of the median nerve. (See Pain—Causalgia, p. 423.)

(v) *Ulnar nerve.*—The ulnar nerve supplies the flexor carpi ulnaris and the greater part of the flexor digitorum profundus in the forearm, and most of the intrinsic muscles of the hand (Fig. 120). Its sensory distribution is indicated in Fig. 121. The deformity caused by ulnar palsy is usually obvious (Fig. 122), and is due to paralysis of the intrinsic muscles of the hand, especially those of the little and ring fingers. The main result of the motor loss is an inability to extend the interphalangeal joints and weakness of flexion of the metacarpophalangeal joints of the ring and little fingers so that the patient has difficulty in getting these digits round objects which he wishes to hold in the palm of the hand. The finer and more delicate movements of the hand are abolished, and there is weakness of lateral movements at the metacarpophalangeal joints. Nevertheless, some lateral movement can often be carried out by trick movements except in the case of the middle finger. There is weakness of active adduction of the thumb, and attempts to perform this movement are accompanied by flexion of the interphalangeal joint. In old-standing cases marked wasting of the interossei is typical. The sensory loss (Fig. 121) is not as disabling as that experienced in lesions of the median nerve. The clinical picture must be distinguished from that of a lesion of the lower roots of the brachial *Deformity*
Motor loss
Sensory loss
Differential diagnosis

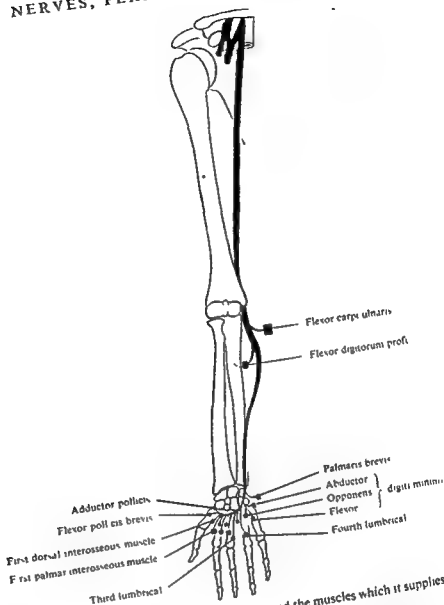


FIG 120—Diagram of the ulnar nerve and the muscles which it supplies.

plexus, such as the cervical rib syndrome, in which all the intrinsic muscles tend to be affected and there may be sensory loss along the medial border of the forearm.

Traumatic ulnar neuritis is a chronic compression lesion of the ulnar nerve in the region of the medial epicondyle of the humerus. Owing to its anatomical position the nerve is particularly subject to repeated trauma at this level, especially if the fascial band which normally retains it in the groove on the back of the epicondyle is slack, so permitting undue lateral mobility of the nerve. Under such circumstances any additional occupational factor is liable to set up a neuritis. In other cases the condition follows a fracture of the medial epicondyle and is then due to friction of the nerve against the roughened bone at the site of fracture. Cases of fracture of the lateral condyle of the humerus, which have healed with proximal displacement of the fragment and consequent valgus deformity of the elbow, are liable to be followed after

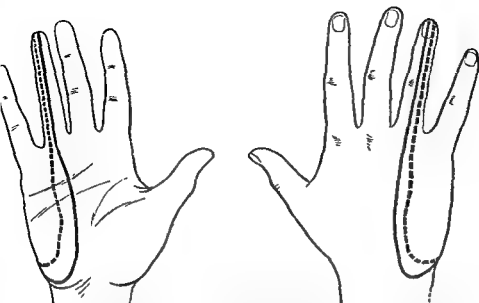


FIG. 121 —The area within which sensory changes may be found in high lesions of the ulnar nerve. In lesions at the wrist the dorsal branch generally escapes and there is no sensory loss on the dorsum of the hand. Light touch, heavy line. Pin prick, broken line.

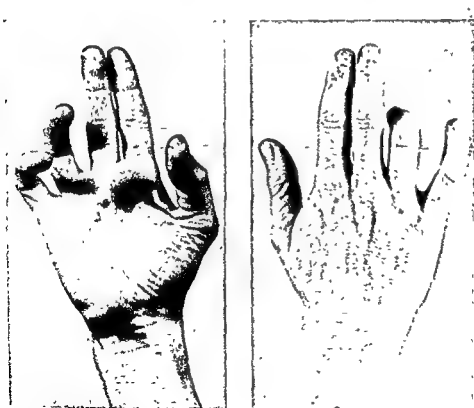


FIG. 122 —Deformity in a case of recent injury to the ulnar nerve. There is clawing of the ring and little fingers and a tendency for the little finger to stand away from the ring finger.

Symptoms
Clinical
features

Treatment

Prognosis

many years by traumatic ulnar neuritis because of the nerve being unduly stretched round the medial epicondyle during movements of the joint (tardy ulnar palsy). The patient's attention may first be drawn to the condition by sensory disturbance in the ulnar distribution, weakness of the hand or wasting of the interossei, and all three symptoms may be present together. On examination it is often possible to feel a "neuroma" on the nerve at the level of the epicondyle, and there is evidence of a partial or complete interruption of conduction in the nerve. Frequently the wasting of the intrinsic muscles is greater than one would expect from the degree of paralysis. As soon as the condition is diagnosed the nerve should be transposed to the front of the elbow, placing it deep to the muscles arising from the common flexor origin (see p. 247). The degree of recovery which may be expected depends largely on the duration of the neuritis and upon the severity of muscle wasting, but at least the operation prevents the condition from getting worse.

(vi) *Median and ulnar nerves combined.*—In combined lesions of the median and ulnar nerves, there is paralytic clawing of all the fingers, and the thumb

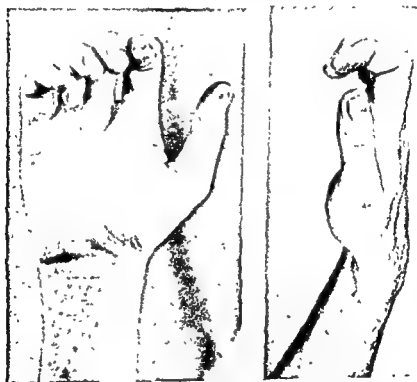


FIG. 123.—Type of deformity seen in cases of combined median and ulnar lesions, especially when the lesions are at the wrist. There is clawing of all the fingers and the thumb lies in the plane of the palm.

Essential
notes

is held in the simian position (Fig. 123). The condition is to be distinguished from Volkmann's contracture of the flexor group of muscles in the forearm, but the two conditions may coexist.

(b) *Lower limb*

ponents

(i) *Sciatic nerve.*—The sciatic nerve in the gluteal region and thigh is really a combination of two nerve trunks—the medial and the lateral popliteal—

enclosed within a single sheath. The two nerves are in fact sometimes separate entities throughout their whole length from the origin in the lumbo-sacral plexus. Consequently, lesions of the sciatic nerve can be regarded as lesions of one or both of the two components even in the thigh or gluteal region. Complete lesions of both components cause paralysis of all muscles below the knee. If the lesion is high up in the thigh or in the gluteal region, there will also be paralysis of some of the hamstring muscles, but it is unusual for all these to be affected, as some of them are supplied by branches which arise

Motor and sensory loss

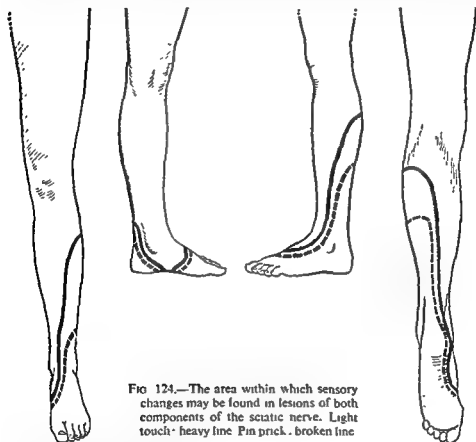


FIG. 124.—The area within which sensory changes may be found in lesions of both components of the sciatic nerve. Light touch—heavy line Pin prick—broken line

very high up in the gluteal region. On the sensory side there is also cutaneous anaesthesia and analgesia, as depicted in Fig. 124.

Partial lesions of the sciatic nerve are occasionally accompanied by the *Causalgia* severe burning pain in the foot known as *causalgia*. (See Pain—Causalgia, p. 423.)

(ii) *Medial popliteal nerve (including posterior tibial).*—The motor and sensory distributions of this nerve are indicated in Figs. 125 and 126. Loss of motor power affects all the muscles in the posterior compartment of the leg and all the intrinsic muscles of the sole. If the lateral popliteal nerve is intact the patient develops a *pes calcaneus* deformity with a tendency to clawing of the toes. The latter deformity is especially noticeable in lesions of the posterior tibial nerve below the origin of the muscular branches in the leg owing to paralysis of the intrinsic muscles and the unopposed action of the

Deformity

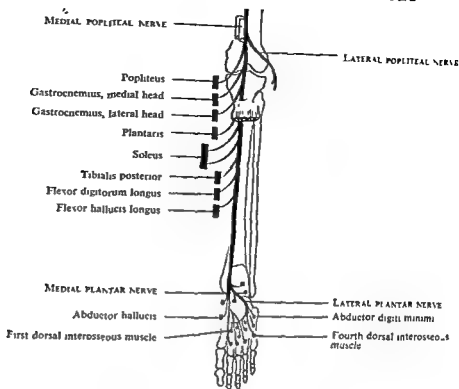


FIG. 125—Diagram of the medial popliteal nerve and the muscles which it supplies



FIG. 126—The area within which sensory changes may be found in lesions of the medial popliteal or posterior tibial nerves. Light touch: heavy line. Pin prick: broken line

Disability

long flexors and extensors. The disability from the paralysis is considerable, especially if the calf muscles are involved. The patient has a pronounced limp, due to difficulty in taking off from the affected foot. This part of the disability is less obvious if there is some contracture of the calf muscles. Some active plantar flexion of the ankle may be retained, the movement being carried out by the peronei. The loss of sensation in the sole of the foot renders the skin liable to develop trophic sores.

Trick movement

Motor loss

(iii) *Lateral popliteal nerve.*—This nerve supplies the muscles of the anterior and lateral compartments of the leg and the short extensors of the toes (Fig. 127). Loss of motor function causes foot-drop with a tendency to inversion. There is paralysis of all the dorsiflexors of the ankle and evertors of the foot.

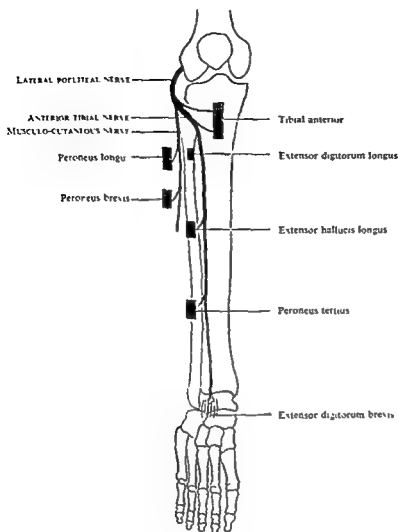
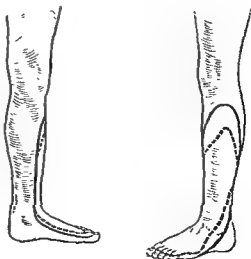


FIG. 127.—Diagram of the lateral popliteal nerve and the muscles which it supplies.

FIG. 128.—The area within which sensory changes may be found in lesions of the lateral popliteal nerve. The sensory loss is often confined to the dorsum of the foot. Light touch: heavy line. Pin prick: broken line.



Sensory loss

and there is an inability to extend the toes at the metacarpo-phalangeal joints. The extent of sensory loss varies according to whether or not the sural nerve is involved, and is frequently less than that depicted in Fig. 128, even in complete lesions. The loss of sensation is unimportant from the point of view of function.

Motor and sensory loss

(iv) *Femoral nerve*.—Lesions of the femoral nerve are not common, but may occur as a complication of wounds of the groin or lower abdomen. There is paralysis or weakness of the quadriceps which naturally constitutes a serious disability. Fortunately the lesion is usually incomplete and in most cases a considerable degree of spontaneous recovery takes place. There is some loss of sensation on the front of the thigh.

6. SPECIAL AIDS TO DIAGNOSIS

In the majority of cases of injury to the peripheral nerves special aids to diagnosis are unnecessary. Careful clinical examination, followed if necessary by an exploratory operation which is regarded as part of the diagnostic procedure, usually gives all the information required. In some cases, however, certain investigations may be of assistance both in determining the type of nerve lesion present and in indicating the prognosis.

(1) Electrical reactions

(See Electrical Reactions of Muscle and Nerve in Vol. 3, p. 350.)

Reaction of degeneration

Although much more accurate information regarding the electrical responses of muscles can be obtained by using an electronic stimulator which measures both the duration of the stimulus and the strength of the current (Ritchie, 1944), it is realized that only the common type of galvanic and faradic stimulator will usually be available. As the motor nerves to a muscle undergo degeneration following division of, or severe damage to, the axons at a higher level, the reaction of degeneration develops in the muscle. It no longer responds to faradism and the response to galvanism becomes slow and worm-like. It is important to realize that, even when there has been complete division of a nerve, a reaction of degeneration takes 2 or 3 weeks to develop, so that the electrical reactions are of little help in distinguishing between a lesion resulting in peripheral degeneration and a mere transient physiological block during the early days after injury. During recovery, too, the electrical reactions, as tested by the ordinary type of stimulator, are of little value, for weak voluntary power is nearly always evident before the response to faradism (percutaneous) returns. If, however, the faradic current is applied directly to the nerve trunk at operation a response can be obtained in the muscles some weeks before the appearance of voluntary power, and this fact can occasionally be used in attempting to assess nerve function at operations done at a certain stage. The response to faradism is also of value in the differential diagnosis between an organic nerve lesion and a hysterical palsy.

Limitations

(2) Sweating test

Method

This test is occasionally of use when definite evidence is required regarding conductivity in the autonomic (sudomotor) fibres. The affected limb is dusted with quinizarin powder, and the other limbs are immersed in hot water until the patient sweats profusely. The sweat changes the pale mauve colour of the

dry powder to a deep purple, and a photographic record of the anhidrotic area can be obtained. The test is also of use in distinguishing between an organic and a hysterical anaesthesia.

(3) Diagnostic nerve blocks

Although temporary blocking of nerves with procaine has its main application in the pre-operative assessment of the results to be expected from sympathectomy, it is also of use in some of the more difficult cases of injury *Uses* to the peripheral nerves, especially when an abnormal innervation is suspected (Highet, 1942).

In dealing with the main nerve trunks of the limbs the aim should be to *Method* inject beneath the nerve sheath a 2 per cent solution of procaine in normal saline. Blockage of conductivity in all fibres should occur within a few minutes. In the case of smaller nerves, such as the digital branches, a perineural infiltration is generally effective.

(4) Radiography

Radiography may be of use if there is a retained radio-opaque foreign body, in that it will indicate the direction of the wound track. It is, of course, also used when a nerve lesion is thought to be due to a fracture or dislocation. The use of radiography in detecting post-operative rupture at a nerve suture line is described on page 246.

7. DIFFERENTIAL DIAGNOSIS

The following are the main conditions which may occasionally be mistaken for a nerve injury.

- (1) Peripheral neuritis (atraumatic).
- (2) Diseases of the central nervous system, such as disseminated sclerosis, syringomyelia and anterior poliomyelitis.
- (3) Myopathies.
- (4) Other conditions of the muscles and fasciae giving rise to deformities, such as Volkmann's contracture and Dupuytren's contracture
- (5) Hysterical palsy and anaesthesia.
- (6) Malingering.

Hysterical conditions and malingering sometimes give rise to difficulties in diagnosis, especially when they are superimposed on a transient organic nerve lesion. In such cases, the apparently paralysed muscles are not grouped according to the normal nerve supply but according to certain movements, so that all muscles involved in such movement, whatever their nerve supply, appear to be paralysed. The muscles do not show the reaction of degeneration, and there is little if any wasting. Furthermore, the patient can often be tricked into contracting an apparently paralysed muscle by persuading him to perform a movement which will normally bring the muscle into play as a synergist. The anaesthesia is generally of the glove or stocking type, and does not correspond to the distribution of one or more of the peripheral nerves. Sweating is not abolished in the anaesthetic area. It is, however, most important that in such cases the possibility of an organic lesion should be excluded by careful examination before a diagnosis of hysteria is made. The type of nerve lesion most frequently mistaken for hysteria is that due to ischaemia of the nerve trunks, for in such cases there is a tendency for the paralysis to

Hysteria and malingering

Importance of careful examination

affect all the more distal muscles and for the anaesthesia to be of the glove or stocking type.

8. PROGNOSIS

(1) Neurological recovery

Neurological recovery in any case of peripheral nerve injury depends on many factors, the most important being the type of nerve lesion present. For example, a nerve affected by a transient physiological block may be expected to have recovered completely within 2 or 3 months, whereas a nerve which has been divided and sutured at a high level may not reach the limit of its recovery for as long as 3 years, and the amount of recovery may be so small as to have little effect on function. The following paragraphs indicate the degree of recovery to be expected in the various types of nerve lesion.

(a) Anatomical division

No appreciable recovery can be expected after such division unless a formal nerve suture has been performed. The degree of neurological recovery following nerve suture depends on the nerve involved, the level of the suture, the time interval between injury and suture, the extent of the injury and, to some degree, the age and general condition of the patient. The radial nerve is by far the most satisfactory, and it is not unusual for almost full voluntary power to be regained. Probably the main factor responsible for the good results is that the radial nerve contains a preponderance of motor fibres, and there is much less chance of motor fibres regenerating along sensory channels and vice versa, whereas in a more mixed nerve it is inevitable that, after suture, many motor fibres will eventually reach sensory end organs, and will of course be useless from the point of view of function. The same factor probably accounts for the surprisingly good results sometimes obtained after suture of the various branches of the median and ulnar nerves in the hand, in spite of the technical difficulties involved. No definite rule can be laid down as regards prognosis after suture at different levels. Although a more satisfactory suture can usually

especially as regards the motor functions. A long period of denervation of a muscle allows more wasting and degeneration of the muscle fibres, and a muscle which has been deprived of its nerve supply for more than 2 years cannot be expected to show any appreciable recovery owing to irreversible changes in the motor end plates. Large gaps in the nerve trunks tend to result in a less accurate orientation of the nerve bundles at the suture line, and the production of a superadded traction lesion of the nerve during the post-operative stretching period further reduces the chances of a satisfactory result (Higbet and Holmes, 1943, Higbet and Sanders, 1943). The results of nerve suture are particularly good in children and adolescents.

(b) Compression lesions

When the axons are divided but the nerve sheath remains intact (a lesion in continuity), the prognosis is, on the whole, better than after nerve suture, but it depends very much on the amount of intraneural scarring. When sensation recovers, tactile localization is much more perfect than after suture. In cases

Wide
variation in
different
types of
lesion

The nerve
involved

Level of
suture

Time interval

Large gaps

Age

Axonotmesis

Neurapraxia

of transient physiological block full recovery can be expected in a matter of weeks.

(c) *Traction lesions*

Usually the results are poor especially as regards motor recovery.

(d) *Ischaemic lesions*

In ischaemic lesions, there is considerable variation in the degree of recovery depending on the amount of intraneural fibrosis. The "slow" pain fibres recover more readily than do the other sensory types, and the final result is frequently a condition of painful over-response to sensory stimuli which is extremely unpleasant.

(e) *Chemical lesions*

The prognosis is, on the whole, poor.

(2) Functional recovery

There are many factors, apart from the actual neurological recovery, which are of importance in prognosis. Ultimate function depends so very much on the condition of the joints, muscles and skin, and this will often affect decisions as to treatment. The patient's mental attitude towards his injury, his willingness to co-operate fully during the long period of recovery, and his occupation, have all to be taken into consideration.

9. INDICATIONS FOR SURGICAL INTERVENTION

It may be taken as a general rule that, whenever there is reason to believe that a nerve may have been divided, exploration is indicated as soon as possible after 3 weeks from the time of injury, provided there are no special contra-
indications to operation. Many surgeons advise that, whenever possible, primary suture should be carried out within a few hours of injury but, although this may be safe and convenient in a few cases in which contusion of the tissues is minimal and there is little risk of infection of the wound, the balance of opinion is in favour of early secondary suture. In order to carry out a satisfactory primary suture, it is generally necessary to extend the original wound longitudinally in order to free the nerve, and this exposes undamaged tissues to the risk of infection. Apart from this danger, which may be slight with the use of penicillin, the extent of the damage to the nerve above and below the site of actual section is much easier to assess at a later stage. Furthermore, the thickening of the nerve sheath, which occurs during the first 2 or 3 weeks after injury, makes an accurate suture far easier to perform. There is little doubt, therefore, that an early secondary suture is a much more precise procedure than a primary one. Consequently the aim, at the primary treatment of the wound, should be to obtain closure of the skin and early healing, and it may be advisable to apply a primary split-skin graft for this purpose. If the divided nerve is seen during the initial treatment of the wound, the ends can be loosely approximated with a single stitch to prevent undue retraction and to facilitate suture at a later date when the wound is healed. A definite note to the effect that formal nerve suture has not been performed should be made on the case sheet, especially if subsequent treatment is to be carried out elsewhere. Should tendons also be divided in a wound in which the danger of infection is slight, it is probably best to do a primary tendon

Optimal time for operation

Arguments against primary suture

Primary treatment of the wound

Treatment of divided tendons

suture and to suture the nerves at a second operation after the tendons have begun to act. If this procedure is adopted, there is less chance of the nerve suture line becoming firmly adherent to the tendons, a state of affairs which may give rise to pain.

*Early
exploration*

*Delayed
exploration*

*Examples of
time of
spontaneous
recovery*

The cases requiring exploration as soon as possible after 3 weeks from the time of injury are those with an open wound in the vicinity of a nerve trunk with signs of complete loss of conductivity in the nerve. In the case of closed injuries, such as crush injuries and simple fractures, it is often justifiable to delay exploration even when there are signs of complete loss of conductivity or of peripheral degeneration. A transient physiological block will recover within 5 or 6 weeks; if peripheral degeneration occurs spontaneous recovery will be delayed for from 3 to 12 months, the period depending on the level of the lesion (Seddon, Medawar and Smith, 1943). The following examples of the latter type of case show how long it may be necessary to wait before the first signs of recovery become evident.

In axonotmesis of the radial nerve, complicating fracture of the middle third of the shaft of the humerus, it takes about 5 months before there are signs of commencing recovery in the brachioradialis.

In a traction lesion of the fifth cervical nerve root the deltoid takes about 9 months to show signs of commencing recovery.

In axonotmesis of the lateral popliteal nerve, at the level of the neck of the fibula, it takes about 5 months for signs of recovery to appear in the tibialis anterior and peroneus longus.

*Late
exploration*

In such cases, when expectant treatment is adopted in the hope that regeneration is taking place, it will generally be necessary to rely on clinical examination for the first signs of recovery, unless an electronic stimulator (Ritchie, 1944) or electromyogram is available. If there is no evidence of recovery within a reasonable time the nerve should be explored, and it is in these circumstances that direct electrical stimulation of the nerve at operation may yield useful information regarding recovery. In any case in which there is doubt as to whether exploration is indicated, it should be borne in mind that, with reasonable care, there is nothing to be lost by exploring.

*Unreliability
of Tinel's
sign*

A warning should be given about regarding a positive Tinel's sign as evidence that satisfactory recovery is taking place. The sign is elicited by lightly percussing over the line of the nerve from the periphery towards the lesion. The sign is positive when, at a point still distal to the level of the lesion, paraesthesiae are felt by the patient in the cutaneous distribution of the nerve. The subjective sensation is due to stimulation of the growing ends of the regenerating axons, but it may be positive in cases of complete division of the nerve in which a few axons have happened to find their way into the distal sheath, but in which there is no hope of useful functional recovery without suture.

10. PRE-OPERATIVE MANAGEMENT AND TREATMENT OF PATIENTS NOT REQUIRING OPERATION

*ning
skin*

Apart from the treatment of the paralysed muscles on the lines indicated below, the pre-operative management will include measures aimed at securing good skin cover in the region of a prospective nerve suture line. Thus dense adherent scars may require excision, and the resulting skin defect may have to

be closed by full-thickness sliding or pedicle grafts. The healing of granulating areas may be hastened by the application of split-skin grafts. In cases in which there is reason to believe that, because of limitation of the range of movement in the joints, there will be difficulty in overcoming the gap in the nerve, it may be necessary to delay operation so that an attempt can be made to mobilize the joints. *Overcome joint stiffness*

In all cases of peripheral nerve injury it is of the utmost importance that the affected muscles, tendons, joints and skin be maintained in the best possible condition so that, if and when recovery of the nerve takes place, the maximal return of function can be obtained. Thus constant care and attention on the part of the physiotherapist is essential, often for many months, and occupational therapy, both remedial and diversional, is also of great value in maintaining the patient's morale and aiding functional recovery. Constant overstretching of the paralysed muscles must be avoided, and to this end it is often necessary to adopt some form of splint, especially in the case of those muscles which tend to be stretched by the force of gravity, such as the deltoid, the biceps and the dorsiflexors of wrist and ankle. Splintage does not mean immobilization, and the best type of splint is one that permits movement of the joints—preferably by the patient's own unparalysed muscles—and yet relaxes the paralysed muscles in the resting position. Moreover, the splints should be removed at least once a day to permit all the joints to be put through a full range of passive movement unless, of course, other considerations, such as the treatment of a fracture, preclude this. *Importance of care of denervated structures*
Physiotherapy
Splinting

It has now been demonstrated convincingly (Gutmann and Guttmann, 1944) that stimulation of the paralysed muscles with a galvanic current does much to minimize the inevitable wasting and fibrosis which occur during the period of denervation, but to be effective this treatment must be commenced at an early stage, before wasting has taken place, and it must be adequate in amount. The aim should be to obtain at least 50 strong contractions of each paralysed muscle every day, and unless the physiotherapeutic facilities make this feasible, it is hardly worth while giving galvanism at all. The same remark applies to cases which have not received electrical treatment during the first 3 months after injury. *Joint movements*
Electrical stimulation

The maintenance of a full range of movement in all joints is of paramount importance. Active exercises should be supplemented by passive movements, and early dissipation of oedema by gentle massage and by elevation of the limb is essential if joint stiffness is to be prevented. *Maintenance of joint movements*

Patients must be warned repeatedly of the susceptibility of the anaesthetic skin to injury and especially of the danger of quite moderate degrees of heat applied locally. The physiotherapist, too, must take special care when treating an anaesthetic limb with radiant heat or wax baths. *Care of skin*

11. OPERATIVE TECHNIQUE

Operations on the peripheral nerves are most conveniently carried out under general anaesthesia and with the limb exsanguinated, but there are certain advantages in using local infiltration anaesthesia, especially when it is doubtful whether or not the nerve has been divided. For local infiltration anaesthesia, a freshly made $\frac{1}{2}$ per cent solution of procaine, containing 1 part in 200,000 of adrenaline, is recommended. Care is taken to avoid injecting the *Anaesthesia and haemostasis*
Local anaesthesia

solution into the nerve sheath, as it is not desired to block conduction in the nerve. Consequently, after making a subcutaneous infiltration in the line of the proposed skin incision, the deeper structures are anaesthetized by inserting a long needle through the skin over the line of the nerve at intervals along the length of the proposed field and into the deeper structures on either side of the nerve by directing the needle always at an angle of 45 degrees to the line of the nerve (Fig. 129) It is advisable to anaesthetize the whole of the

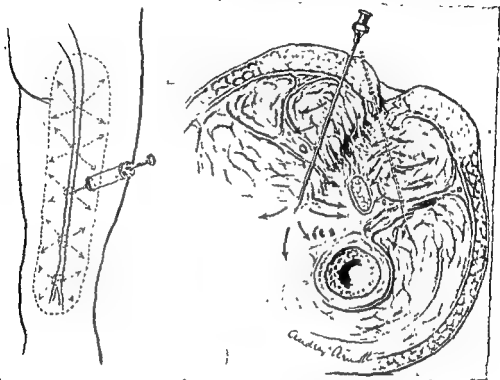


FIG. 129.—Diagram to illustrate the method of infiltrating the deep tissues with local anaesthetic. The aim is to avoid blocking the nerve trunk itself.

*Method of
haemostasis*

proposed field before commencing the operation. When a general anaesthetic is used the limb is exsanguinated by applying an Esmarch bandage from below upwards, and then applying a sphygmomanometer cuff or pneumatic tourniquet and inflating the latter to well above the systolic blood-pressure before removing the Esmarch bandage. Naturally such a tourniquet cannot be used when the operation field is to be extended to the proximal part of the limb, and it is neither necessary nor advisable when using local infiltration anaesthesia.

*Tourniquet
palsy*

There is little danger of harm resulting from the use of a pneumatic tourniquet as described above, but no other type of tourniquet should be used on the upper limb as there is a considerable risk of producing a tourniquet palsy. Such a nerve lesion is nearly always of the compression type, caused at the time of application of the tourniquet, and is unrelated to the length of time for which the tourniquet is in position. There may, however, be some generalized ischaemic damage if the limb is continuously deprived of all blood supply for more than 1½ hours.

As a general rule the skin incision is made immediately over the line of the nerve, but its direction may, with advantage, deviate from this line in certain areas, in order to excise scar tissue, to overlap intermuscular intervals or to avoid crossing at right angles to natural skin creases in the region of joints. The incision should be of adequate length and should extend at least for some distance above and below the level of the suspected lesion. It is important to begin by exposing the nerve above and below the damaged area where the normal anatomy is not distorted by scar tissue. The nerve is therefore isolated on either side of the lesion, and moist tapes are placed round it and secured in pressure forceps so that it can be controlled with the minimum of trauma. The nerve is then traced towards the lesion, care being taken not to damage either the main trunk or any important branches. If there is much scar tissue, this may be a long and tedious procedure requiring considerable patience on the part of the surgeon.

(1) Exposure of individual nerves

(a) Radial nerve

The method of approach depends very much on the level of the suspected lesion. In high lesions near the axilla the nerve is best approached through an incision over the line of the main neurovascular bundle, that is over the medial border of the biceps, dividing the lower fibres of the pectoralis major close to their insertion, if necessary. The biceps and the musculo-cutaneous nerve are retracted laterally. The brachial vessels and the median and ulnar nerves are identified and retracted medially with a moist tape. The large radial nerve is identified where it lies on the tendon of latissimus dorsi, and from that level can be traced down to the point at which it enters the radial groove of the humerus. In lesions below the middle of the arm the incision is made over the interval between the brachioradialis and brachialis muscles, and the nerve is exposed as it lies deeply in the groove between these two muscles. From here it can be traced up to the lower end of the radial groove, the lateral head of the triceps being divided if necessary, and down to the level of the neck of the radius where it divides into the superficial radial and posterior interosseous branches. The posterior interosseous nerve can be exposed for about $1\frac{1}{2}$ inches below this by dividing the superficial fibres of the supinator muscle. Incidentally, suture of this nerve usually gives excellent results. In lesions situated at about the middle of the arm, it may be more convenient to expose the nerve from the back by an incision over the triceps and by opening up the interval between the long and lateral heads of that muscle.

(b) Musculo-cutaneous nerve

The skin incision is made over the medial border of biceps below the axilla. The biceps is retracted laterally and the lower fibres of pectoralis major may need to be divided close to their insertion. The nerve is exposed at a point lateral to the main neurovascular bundle above or below the point at which it pierces coraco-brachialis.

(c) Median Nerve

In the upper arm the nerve is exposed through an incision over the medial border of the biceps. The muscle is retracted laterally and the nerve, which is easily recognized by its large size, is found lying with the brachial vessels.

Elbow

At the elbow it is advisable to make the skin incision with an S-shaped configuration to minimize the danger of producing a keloid scar. The median nerve lies in the antecubital fossa medial to the tendon of the biceps and beneath the bicipital fascia, and the several branches which it gives to the pronator teres and the long flexor muscles in the forearm must be carefully preserved.

Forearm

In the forearm a median skin incision is used. The median nerve passes down under cover of the pronator teres, flexor carpi radialis and flexor digitorum sublimis, and tends to adhere to the deep aspect of these muscles, especially to the last-named the radial head of which will require to be divided.

Wrist

At the wrist the skin incision should be made with an S-shaped curve to minimize the risk of keloid formation in the scar. The nerve lies immediately beneath the deep fascia on the ulnar side of the tendon of flexor carpi radialis until it enters the carpal tunnel in which it lies deeply beneath the flexor retinaculum. It is generally necessary to divide the retinaculum in order to secure the distal portion of the nerve after traumatic division at the wrist.

In the palm
Danger of
damaging
the motor
branch

In the palm the branches of the median nerve can be exposed through an incision in the "life line". Very great care is required to prevent accidental damage to the motor branch to the thenar muscles which hooks round the distal border of the flexor retinaculum and becomes very superficial where it runs in a lateral and often proximal (recurrent) direction beneath the thin layer of fascia covering the origin of the thenar muscles before entering the latter. At this point the motor branch is often thin and translucent and can easily be mistaken for a strand of fascia.

(d) *Ulnar nerve**Upper arm*

In the upper arm the skin incision is made directly over the line of the nerve from the point at which the main neurovascular bundle emerges from beneath the lower border of pectoralis major to the interval between the medial epicondyle of the humerus and the tip of the olecranon. In the lower half of the upper arm the nerve lies immediately behind the medial intermuscular septum.

Forearm

In the forearm the skin incision is made again over the line of the nerve from just behind the medial epicondyle to the radial side of the pisiform bone. In the upper half of the forearm it is necessary to split the fibres of flexor carpi ulnaris to expose the nerve lying beneath the fascia covering flexor digitorum profundus, whereas in the lower half of the forearm flexor carpi ulnaris can be retracted backwards.

The palm

In the palm, the skin incision can be continued down over the hypothenar eminence, where the nerve and its branches lie deep to the lobulated superficial fat found in this region. When clearing the nerve care is necessary to avoid damaging the deep motor branch which arises from its deep aspect just distal to the hook of the hamate bone.

(e) *Sciatic nerve**The buttock*

In the buttock a curved incision, convex laterally, is made with the summit of the curve just medial to the greater trochanter of the femur so that the gluteus maximus can be divided fairly close to its insertion and reflected medially to expose the nerve.

In the thigh the skin incision is made in the posterior midline and the long head of the biceps femoris is retracted medially or laterally depending on whether the nerve is to be exposed in the upper or lower half of the thigh. *The thigh*

(f) *Medial and lateral popliteal nerves*

These two main divisions of the sciatic nerve are easily exposed in the popliteal space. The lateral popliteal nerve becomes quite superficial at the level of the knee joint, and can be followed round the neck of the fibula to the point where it divides into the anterior tibial and musculo-cutaneous nerves. *The popliteal space*

(g) *Posterior tibial nerve*

In the leg this nerve is probably most easily exposed through a posterior midline incision, splitting the division between the two heads of the gastrocnemius muscle and dividing the bipennate fibres of the soleus muscle. The nerve lies very deeply beneath the latter muscle. *The leg*

At the ankle, the nerve and its terminal branches—the plantar nerves—lie beneath the flexor retinaculum and can easily be exposed through an incision curving behind and below the medial malleolus. *The ankle*

(2) *Subsequent procedure*

When the damaged nerve has been separated from the surrounding structures and from scar, it may be quite obvious that it has suffered complete division. More often, however, the undamaged parts of the nerve are connected by a mass of tissue which may be purely scar or may contain intact nerve fibres. In other cases, the nerve sheath may be intact although there may be signs of intraneural damage. Except in cases in which the nerve has obviously been completely divided, the main concern of the surgeon is to decide whether or not resection of the damaged portion and suture should be carried out, and this is frequently a matter of considerable difficulty. The presence of a firm bulbous neuroma at the point where the nerve enters the damaged area indicates that at least some of the fibres have been divided; this is a point in favour of resection and suture. A soft fusiform neuroma inside an intact sheath is, however, usually present with a lesion in continuity and is not an indication for suture. When the nerve has been divided there is usually a small glioma where the nerve leaves the damaged area. The consistency of the nerve is carefully estimated by palpation. An area of dense induration, especially below a neuroma, is an indication for suture. It may sometimes be difficult to decide, however, whether the scar is intraneural or merely attached to the outside of the nerve sheath. A narrow waist below a neuromatous enlargement is usually an indication for suture, but if some structure such as a fragment of bone is observed to have been constricting the nerve at this level, and if there is little induration, the nerve may well recover more completely without suture. *Estimation of nerve damage*, *Neuroma*, *Intraneural scarring*, *Constriction*

In cases of doubt, the nerve may be stimulated directly with a faradic current preferably using a bipolar electrode. This procedure gives much more information when the operation is being performed under local anaesthesia, because the conduction of sensory impulses across the damaged area can then be tested, provided the local infiltration has been carried out, as previously described, in order to avoid blocking conduction in the nerve. Before applying electrical stimuli it is advisable to remove any tourniquet. If the nerve is *Electrical stimulation*

insensitive to stimulation below the level of the lesion, whereas the stimulus of the same current above the lesion can be felt, sensory conduction is obviously abolished at the site of the lesion. Similarly, a weak response below and a strong response above the lesion indicate that some of the sensory fibres have been destroyed. If stimulation above the lesion produces a definite contraction in the muscles supplied by the nerve at a lower level, the motor fibres are obviously intact, but the lack of such response does not necessarily mean that the nerve is not recovering spontaneously from a lesion in continuity.

Other considerations

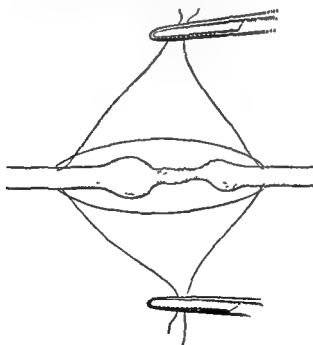
Other factors, such as the length of time since injury, the chances of getting a good result from suture, and the possible disadvantages to the patient of immobilization after suture have all to be taken into account in a difficult case.

The importance of a correct decision cannot be overestimated. The easy way appears to be to wait and see whether the nerve recovers spontaneously before deciding to suture, but by this time the chances of getting a good motor result from suture are often remote.

(a) Technique of suture

The ideal to be aimed at is to resect the whole of the damaged portion of the nerve and to obtain accurate apposition of apparently "healthy" nerve bundles without undue tension at the suture line, and using the minimum of foreign suture material. This ideal is frequently unattainable if more than a short length of the nerve is damaged, because the more one resects in order to get "healthy" nerve, the less chance there is of being able to suture without tension. Although the gap can easily be overcome by freeing the nerve and positioning the joints, there is still the danger of producing a traction lesion

Limiting factor



Stay sutures

FIG. 130—Method of inserting stay sutures.

in the nerve during the post-operative stretching period (Higbet and Holmes, 1943; Higbet and Sanders, 1943). Such stretching, especially if confined to a short length of nerve, has deleterious effects, particularly on the motor fibres. Before resecting the damaged portion of the nerve, it is advisable to insert temporary stay sutures which will assist in preventing axial rotation of the stumps and will be used to control them during the performance of the suture. Fine silk is used, preferably on an eyeless needle; the nerve sheath is picked up some distance on either side of the damaged area. Two such sutures are used, and are inserted as shown in Fig. 130. The nerve trunk is cleanly divided

in the nerve during the post-operative stretching period (Higbet and Holmes, 1943; Higbet and Sanders, 1943). Such stretching, especially if confined to a short length of nerve, has deleterious effects, particularly on the motor fibres. Before resecting the damaged portion of the nerve, it is advisable to insert temporary stay sutures which will assist in preventing axial rotation of the stumps and will be used to control them during the performance of the suture. Fine silk is used, preferably on an eyeless needle; the nerve sheath is picked up

immediately above the obviously damaged area, or immediately above the neuroma if one is present. A new sharp blade is used—a thin safety-razor blade held in a pair of artery forceps is suitable—and, during the section, the nerve is steadied with the stay sutures and if necessary by the fingers of the assistant. The proximal face of the section is then carefully inspected to see whether or not healthy nerve bundles have been exposed. The bundles should be clearly defined, there should be no intraneural scarring, and the nerve sheath should not be unduly adherent to the underlying bundles. If the appearance is unsatisfactory or doubtful, sections of a few millimetres at a time should be made until healthy nerve is exposed, always having regard to the gap to be overcome. Similarly, sections are made below the damaged area where, however, the nerve bundles are generally smaller because of degeneration. An attempt is then made to approximate the clearly divided ends by tightening the stay sutures (Fig. 131). If a gap still remains, or if the ends can be apposed only with difficulty, steps must be taken to overcome the gap. The skin incision is

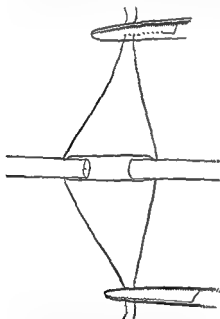
*Resection**Inspection of nerve bundles**Overcoming the gap*

FIG. 131 —The damaged portion of the nerve has been resected and the stay sutures are being tightened

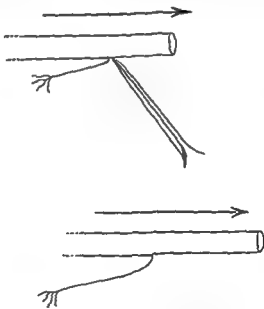


FIG. 132 —Method of stripping up proximal branches to gain extra length

extended, and the nerve is freed from the surrounding structures, care being taken to preserve all important branches. If branches are arising from the nerve above the level of the lesion, and are preventing the nerve from being drawn distally, considerable extra length can often be gained by stripping up the branches by careful sharp dissection (Fig. 132). Extensive freeing of the nerve allows advantage to be taken of its natural elasticity and distributes any post-operative traction over more of its length. Although every effort is made to preserve the segmental blood supply, such stripping has been shown not to cause dangerous ischaemia of the proximal nerve trunk because of the presence of a longitudinal anastomosis within the nerve. The blood supply of

Blood supply

the distal trunk is rather more precarious, and it is unwise to divide any obvious vessels supplying it.

*Positioning
of joints*

Considerable gaps can often be overcome by positioning—that is, generally flexing—the neighbouring joints, especially if the nerve has been previously freed. This is especially applicable to the popliteal nerves at the knee. One should not take too much advantage of this without also freeing the nerve over a considerable length, for fear of producing a traction lesion during the period of subsequent straightening of the joint. Transposition may also be used, where possible, in order to overcome the gap, and is applicable especially to the ulnar nerve at the elbow.

*Danger of
causing
traction lesions*

Suture materials.—The suture materials which cause least tissue reaction, and therefore may be expected to interfere least with nerve regeneration, are very fine silk (unwaxed), stainless-steel wire or tantalum wire. Fine wire is probably best, but it has a greater tendency than silk to cut out of the nerve sheath. An eyeless needle is to be preferred. The sutures are placed at regular intervals round the circumference of the nerve, and pick up the nerve sheath only (Fig. 133). The stay sutures are used to rotate the nerve as shown in Fig. 134. As few sutures are used as is consistent with accurate apposition of

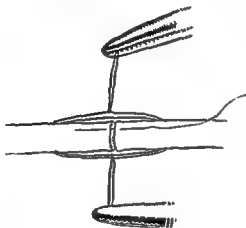


FIG. 133.—The stay sutures have been tied and the first permanent suture is being introduced

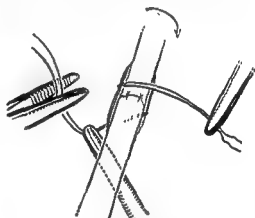


FIG. 134.—Method of using stay sutures to rotate the nerve and expose the deep aspect for insertion of remaining sutures.

*Avoidance of
axial
rotation*

markers

the stumps. Axial rotation must be avoided, and in this connexion it is often possible accurately to match the two faces of section by carefully inspecting the pattern of the nerve bundles. When the suture has been completed, the stay sutures are removed. Radio-opaque markers in the form of small clips, or merely loops of stainless-steel wire, are then placed through the nerve sheath a known distance—say 1 centimetre—on either side of the suture line. Subsequent x-ray examination will then indicate whether or not the suture line has given way. This procedure has proved valuable on several occasions

... months of delay before re-
...
the wound, an attempt should be made to place the sutured nerve in a satisfactory bed remote from scar tissue or separated from it by healthy muscle or fascia.

(b) Transposition of nerves

Considerable extra length can be obtained in certain nerves by transposing them so that they run in a more direct course. This method is especially useful in the case of the ulnar nerve at the elbow, and is often used to overcome even moderate gaps in the forearm. The position of the elbow makes little difference to the length of the ulnar nerve in its normal course, and anterior transposition is preferable to immobilizing the wrist in acute flexion. Anterior transposition is frequently carried out for traumatic ulnar neuritis (see p. 230), in which case there are considerable advantages in placing the nerve close to the median nerve beneath the muscles of the common flexor origin, the latter being divided close to the medial epicondyle and reflected laterally (Learmonth, 1942). When the procedure is used for overcoming a gap in the nerve in the upper arm, it is unnecessary to divide the muscles for, after freeing the distal portion of the nerve, it can be drawn up through a tunnel made by thrusting a pair of long forceps beneath them. Care must be taken to avoid damaging the branches to the forearm muscles which are given off just below the medial epicondyle. The medial intermuscular septum in the upper arm should be removed in order to prevent the nerve from being kinked around it at the upper end of the field. When there is a considerable gap to be overcome, it may be necessary to place the nerve subcutaneously in front of the elbow. It is not advisable to place it in a channel cut in the common flexor origin, because of subsequent dense scar formation round the nerve.

*Technique of
anterior
transposition
of ulnar
nerve*

The median nerve at the elbow can be brought superficial to the muscles of the common flexor origin after stripping up the muscular branches which it gives to the forearm muscles and, if necessary, dividing the pronator teres muscle; by this means considerable extra length can be gained to close gaps in the forearm. The radial nerve is sometimes transposed to the front of the humerus, but in cases of fracture of the shaft of the humerus, which have united with anterior bowing, no gain can be expected from this manoeuvre.

*Other
useful
transpositions*

(c) Nerve grafts

Nerve grafting to overcome gaps in the peripheral nerves has, on the whole, given disappointing results, except in the case of some autografts. Any attempt to bridge a gap by using a length of animal nerve (heterograft) or a nerve from another human subject (homograft) is doomed to failure (Barnes and his colleagues, 1946). Lengths of unimportant cutaneous nerves such as the superficial radial nerve of the forearm or the lateral cutaneous nerve of the thigh can be used with success to replace damaged portions of the digital nerves in the hand. Occasionally, when both median and ulnar nerves have been severely damaged, it is justifiable to use the ulnar nerve as a graft to fill a large gap in the median nerve, preferably by a two-stage operation to preserve the vascularity of the graft (St. Clair Strange, 1947).

*Autografts,
heterografts
and homografts*

(d) Plaster fixation

It is nearly always necessary to apply a plaster to the limb at the conclusion of the operation in order to fix those joints, unrestricted movement of which would cause tension on the suture line. When the sciatic or popliteal nerves have been sutured, this must often take the form of a spica to prevent flexion of the hip and maintain flexion of the knee, unless it can be seen during

operation that flexion of the hip does not put undue tension on the nerve provided the knee is kept flexed.

(e) *Neurolysis*

Sometimes it may be found at operation that the nerve, although not actually divided, is being constricted as, for example, between two bony fragments in the region of a fracture. Provided the sheath is intact and there is no gross intraneural scarring, regeneration will often occur after freeing of the nerve (*neurolysis*). It is extremely doubtful whether extraneural scar tissue can by itself cause such a lesion in continuity, and many patients showing rapid recovery of function following neurolysis from scar would probably have recovered just as quickly without operation. It is not generally appreciated how long such lesions in continuity take to recover spontaneously (*see p. 236*).

(f) *Other operations*

A number of operations are available to improve function when recovery in the nerve is impossible or when adequate follow-up has shown that recovery has come to a standstill, but their description does not come within the scope of this section. The most useful of such operations are arthrodesis

the paralysed intrinsic muscles of the hand are replaced by transplanting the tendons of the flexor sublimis digitorum.

12. POST-OPERATIVE CARE

Removal of plaster

The plaster fixation is maintained for 3 weeks, when the plaster is bivalved and the skin sutures are removed. Gradually controlled extension of the joints is then commenced, the stretching process taking from one to several weeks depending on the degree of flexion of the joint and of tension in the

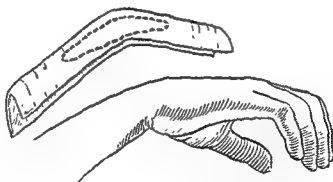


FIG. 135.—Method of decreasing the amount of flexion of the wrist by introducing padding into the angle of a dorsal plaster slab.

Methods of controlling extension

nerve. It is often convenient to retain the convex half of the bivalved plaster, and to obtain controlled extension by introducing padding as shown in Fig. 135, the amount of padding being increased every few days. In the case of the knee, the degree of extension can be controlled by a leather harness as

illustrated in Fig. 136. This has the advantage of permitting active exercises within the limits of extension allowed by the harness.

Skiagrams to show the wire markers introduced at the time of suture should be taken as soon as is convenient after the plaster has been applied and again after the post-operative stretching period. If either of these skiagrams shows separation of the markers, re-exploration should be considered, but in interpreting the skiagrams allowance must be made for the radiographic magnification of the gap between the markers.

The care and treatment of the denervated structures as outlined under Pre-operative Management must, of course, be continued in the post-operative period. Furthermore, the problems of resettlement in a suitable occupation require careful consideration in each individual case. These patients are often most unwilling to use the affected limb, and it is necessary to impress upon them that active exercise is the best way of improving the strength of partially recovered muscles.

13. RESULTS OF SURGICAL TREATMENT

The factors affecting recovery after lesions of the peripheral nerves have been dealt with under Prognosis. It must be admitted that, except in the case of the radial nerve, complete recovery after suture is the exception rather than the rule; if, however, from the time of injury, every attention is given to the care of the denervated structures, the ultimate disability can be reduced to the minimum.

[The illustrations to Figs 112-118, 120, 121, 124-128 are modified from those appearing in *Aids to the Investigation of Peripheral Nerve Injuries* (H.M. Stationery Office)]

REFERENCES

Barnes, R., Bacsich, P., Wyburn, G. M., and Kerr, A. S. (1946). *Brit. J. Surg.*, 34, 34.
Bunnell, S. (1945). *Surgery of the Hand*. Philadelphia and London; Lippincott.
Guthrie, F. (1943). *Brit. J. Surg.*, 30, 355.



Care of denervated structures
Resettlement

FIG 136—Adjustable leather harness to control extension of the knee after removal of plaster.

Learmonth, J. R. (1942). *Surg. Gynec. Obstet.*, 75, 792.

Medical Research Council (1943). *Aids to the Investigation of Peripheral Nerve Injuries*. War Memorandum No. 7. London; H.M. Stationery Office.

Parkes, A. R. (1945). *Brit. J. Surg.*, 32, 403.

Ritchie, A. E. (1944). *Brain*, 67, 314.

Seddon, H. J. (1943). *Brain*, 66, 237.

— Medawar, P. B., and Smith, H. W. (1943). *J. Physiol.*, 102, 191.

St. Clair Strange, F. G. (1947). *Brit. J. Surg.*, 34, 423.

Stiles, H. J., and Forrester-Brown, M. F. (1922). *Treatment of Injuries of the Peripheral Spinal Nerves*. London; Frowde, Hodder and Stoughton.

Zachary, R. B. (1945). *Surg. Gynec. Obstet.*, 81, 213.

[References to other titles are given under Nerves, Peripheral—Injuries, in the Index Volume. The subject is also dealt with under the heading of Nerve Injury and Repair in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 160.]

NEURALGIA—TRIGEMINAL, GLOSSOPHARYNGEAL

By G. F. ROWBOTHAM, B.Sc., F.R.C.S.

SURGEON-IN-CHARGE, NEURO-SURGICAL CENTRE, NEWCASTLE-UPON-TYNE:

LECTURER IN NEURO-SURGERY, UNIVERSITY OF DURHAM

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PART I

PAROXYSMAL TRIGEMINAL NEURALGIA

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1. DEFINITION

243] A diagnosis of paroxysmal trigeminal neuralgia implies that a patient is suffering periodically from sharp shooting pains in the face that are confined to the territory of the trigeminal nerve. These pains are not associated with objective sensory loss in the painful areas and are not due to any kind of pathological change that can be demonstrated either in life or at necropsy.

Synonyms

Other names are often used to designate this disorder; for example, major trigeminal neuralgia, Fothergill's disease, facial tic and tic douloureux.

Distinction from spasmodic tic

It is true that in severe attacks of *paroxysmal trigeminal neuralgia* the facial muscles go into spasm on the affected side, but this is in the nature of a flickering rather than of a full contraction of the facial muscles. Spasmodic tic is a different disease altogether and should not be confused with tic douloureux. Here the primary disorder is excessive spasm of the facial muscles, pain being a secondary concomitant.

The essential features of *spasmodic trigeminal neuralgia* are:

- (1) Periodicity.
- (2) Sharp shooting character of the pain which is confined to the distribution of the trigeminal nerve.
- (3) Absence of objective sensory loss in the face.
- (4) Absence of gross underlying pathological cause.

2. HISTORY

According to Sprengel (1815-20), who is probably one of the most learned of all medical historians, the malady was known to Rhazes and Avicenna. Mueller (1875) stated that the condition was known to Galen. In Wells Cathedral there is a thirteenth-century carving of a face which depicts the agony of a patient suffering the tortures of trigeminal tic. André of France (Gurlt, Wernich, and Hirsch, 1929) and Fothergill of Great Britain in the eighteenth century (1779) were probably the first of the moderns to describe the clinical features of the illness succinctly. Therapeutic advances are attributable to such people as Sir Victor Horsley (1891) and Wilfred Harris (1926 and 1937) of Great Britain, Krause (1892, 1893 and 1896), and Härtel (1912 a and b, and 1929) of Germany, Lévy and Baudouin (1906) of France, and Hartley (1892) and Spiller and Frazier (1901 and 1933) of America.

3. AETIOLOGY AND PATHOLOGY

Aetiology unknown

The aetiology of paroxysmal trigeminal neuralgia is not known. Neuralgia possibly results essentially from the effects of chronic dental sepsis or from degenerative changes in the central nervous system that may occur naturally with advancing age.

Theories of pain causation

The pathology is also unknown. The following are the main theories of the possible causes of the pain:

- (1) Nerve endings for pain become embedded in fibrous tissue so that any movement of the face, however simple, causes drag on the nerve endings and so initiates a painful impulse.
- (2) A low-grade interstitial neuritis may cause faults in conduction so that non-painful modalities of sensation may take on the nature of a painful impulse.

- (4) Misinterpretation of the neural message at the highest level.

(5) Beating of a hardened artery against the root of the trigeminal nerve as it traverses the posterior fossa from the Gasserian ganglion to the pons varolii.

(6) The pain may arise in the wall of a blood-vessel and be aroused by excessive spasm or dilatation of that vessel, the impulse of pain being merely transmitted by the trigeminal pathways.

4. CLINICAL FEATURES

As a rule the pain more or less equally affects men and women who have passed the age of 45 years. Although it occurs equally on either the right or the left side of the face, it has a definite predilection for the mandibular and maxillary branches of the trigeminal nerve. Usually the pain is restricted to one side and remains restricted to this side. In from 1 to 5 per cent of cases (of my series) the pain was bilateral. In bilateral cases the pain very rarely commences simultaneously on both sides; that on the second side usually occurs months or years after the first side has been affected. The pain commonly originates from a localized area known as the trigger-point. Four common sites for these pains to originate in are: the outer half of the lower lip, the side of the tongue, the naso-labial fold and the outer side of the eyebrow. The pain shoots from the trigger-point along the distribution of the nerve concerned like a flash, and is often described as resembling an electric shock or a red-hot corkscrew being driven into the face. *Pain usually unilateral*

Pain starting in the lower lip and lower jaw will radiate upwards towards the lower part of the temple just in front of the ear. Pain starting in the naso-labial fold shoots upwards towards the forehead; occasionally it passes to the outside of the orbit but more usually it radiates to the inside of the orbit on the side of the nose. Pain starting anywhere in the face or lower jaw may radiate to another branch of the nerve or to every limit of the trigeminal distribution. Pain radiating to the vertex of the head means that the ophthalmic division itself is affected. Very rarely does the pain start in the ophthalmic division, but when it does the possibility of other conditions, such as aneurysms of the internal carotid artery, being the cause, must be seriously considered. *Distribution of pain*

The pain, as stated above, occurs in flashes of great severity but is soon over. Such flashes may occur in a severe case every few minutes and such episodes may continue for a day or for weeks. There are, however, periods of complete freedom; these periods may last for many years, although there is a great tendency to recurrence at some time in the future. Often the pain impulse is initiated by a simple disturbance of the face, such as occurs in speaking or chewing or by a light touch with a finger or handkerchief. During a bout of pain, the patient stands motionless, keeping his head and face as still as possible, with his hand to his face but not touching it, as if to ward off any further kind of touch or interference. He never rubs the painful area as patients with other kinds of neuralgia are wont to do. This loathing to touch the face is a sign of great diagnostic importance. In an attack of pain the facial muscles often flutter in a mild spasm; the conjunctivae may become suffused and tears flow. Rarely, the jaws clasp as if in an attack of epilepsy. At the end of an attack the patient is usually completely free of pain and is left without discomfort; occasionally, on the other hand, the face appears to *Nature of pain*
Important diagnostic sign

be alive with pain even during the intermissions. Often, of course, there is deep apprehension that another attack will occur.

On examination, no objective loss of sensation is present in the face and no pathological lesion can be found in the face, in the central nervous system or elsewhere in the body to account for the neuralgia.

5. DIFFERENTIAL DIAGNOSIS

The sharp shooting pains characteristic of paroxysmal trigeminal neuralgia can be very closely mimicked by the pains that arise from the lesions of injury, infection and new growth in the face. From this pathological view-point it is necessary to mention injury of the Gasserian ganglion or of its large divisions; septic and toxic interstitial neuritis; carcinomatous deposits in the sheath of the ganglion; carcinoma of the tongue and antrum; neuromas in the cerebello-pontine angle; aneurysms of the basilar and internal carotid arteries and lesions of the central nervous system, of which disseminated sclerosis is the chief. Indeed, the diagnosis of paroxysmal trigeminal neuralgia should not be considered until the possibility of the presence of each of the above lesions has been reviewed and eliminated by careful examination or special investigation.

The following conditions merit special consideration.

(1) Causalgia of the face

Causalgia may result from closed as well as from open injury of any part of the trigeminal nerve. In this condition the essential diagnostic features are the history of injury, a complaint of continuous discomfort in the face and some degree of objective sensory loss in the painful area.

(2) Interstitial neuritis

there may be present or past evidence of sepsis in the face.

(3) Migraine of the face

Occasionally an attack of migraine may start below the eye or in the eye and spread to the forehead or, as is more usual, the pain may start in the forehead

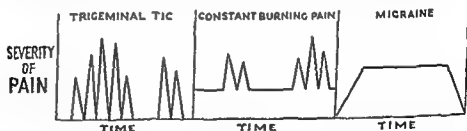


FIG. 137.—A graphic representation of different types of pain.

and spread to the upper cheek. Little difficulty is encountered in recognizing the nature of this kind of pain because other phenomena, such as general malaise, are present. On the other hand, supra-orbital migrainous neuralgia is very difficult to differentiate from paroxysmal trigeminal neuralgia. It is not known whether this condition is, in fact, a true paroxysmal trigeminal neuralgia or whether it is a form of migraine. In my opinion it is migraine. The pain

*Differentiation
from supra-
orbital
migrainous
neuralgia*

is periodic, is very severe, is sharp and shooting and occurs strictly in the distribution of the supra-orbital nerve. However, it is a pain which commonly occurs in patients under the age of 50 and is often associated with other signs of migraine, such as malaise and change in colour. Moreover, the periods of pain are much more prolonged than are the typical flashes of paroxysmal trigeminal neuralgia and they occur primarily in the ophthalmic division of the trigeminus, which is unusual in paroxysmal trigeminal neuralgia. (See graph in Fig. 137.)

(4) Herpes zoster—post-herpetic neuralgia

In post-herpetic neuralgia there are often paroxysms of sharp shooting pain. However, there is always a background of stinging and burning and the skin is hypersensitive to light touch. The diagnosis is obvious when the skin shows the scars of previous vesiculation. Also the ophthalmic division is the one most usually affected.

(5) Continuous burning pains in the trigeminal area

These pains are the most important from the differential diagnosis point of view. They are one of the great enigmas of facial pain. It is of fundamental importance to recognize them because any interference with the trigeminal pathways, either by alcohol block or by severance of the nerve trunk, will make the patient's condition worse rather than better.

Typically, the pain starts in people under the age of 30, although it may occur at any age. Often it follows extraction of a tooth. Immediately after the extraction the patient begins to complain of pain at the site of operation. The pain is burning and stinging in character. It is continuous although there may be periods of sharp shooting stabs. The patient does not resent pressure over the painful area and, indeed, is often seen pressing on it with his own fingers; also he tends to work his jaws incessantly in chewing movements in an attempt to get relief. There is never any sensory change apart, perhaps, from a local area of hyperaesthesia at the site of the pain. There are no signs of local sepsis and none of organic disease in the central nervous system. Possibly the pain is due to a low-grade interstitial neuritis or parasympathetic ganglionitis. On the other hand, these pains are often associated with psychogenic disturbances. Two patients whom I have observed over the last 15 years have both ended in a mental home—one with frank schizophrenia and the other with a delusional insanity. In the early stages the patient often loses weight as in anxiety states. Whether a patient complaining of these burning pains is suffering from a psychalgia, that is, from pains that arise in the mind, or whether he is merely driven mad by the persistency of their discomfort is not known. What I wish to stress is that these pains should be recognized and differentiated from paroxysmal trigeminal neuralgia and faulty treatment avoided. Even tractotomy fails to relieve the pain and leaves the patient with something more of which to complain.

*Possible
psychogenic
associations*

6. TREATMENT

There is no known cure for paroxysmal trigeminal neuralgia. Moreover, the condition does not tend to burn itself out by natural processes. Although intermissions are usual, there is a tendency for the attacks to become more

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frequent later in life, for them to last longer and for the pain to become more severe and more widely spread.

There are three ways of obtaining relief—by medical measures, by alcohol block and by surgical severance of some part of the pain pathways.

(1) Medical measures

Two groups of drugs may be used to give relief from pain, the narcotics and the analgesics. If a narcotic drug is chosen, enough should be given to put the patient quietly to sleep. Patients differ enormously in their reaction to analgesic drugs, and it is only by trial and error that the most suitable one is found for the patient concerned. It is my experience that the time-honoured mixture of tincture of gelsemium and butylchloral hydrate has no particular merit. Whiffs of trichlorethylene will occasionally give dramatic relief in a severe attack of neuralgia. Extraction of the teeth rarely has any effect on the pain; indeed, many of the patients who suffer from this disorder are already edentulous. There appears to be no seasonal incidence for the attacks and changes of climate may or may not lessen the patient's liability to pain.

(2) Alcohol injection

Details of the various injections will be found in Figs. 138, 139 and 140. Painful impulses can be blocked by injecting alcohol into the Gasserian ganglion, into the maxillary and mandibular divisions of the trigeminal and also into the supra-orbital, infra-orbital and inferior dental nerves.

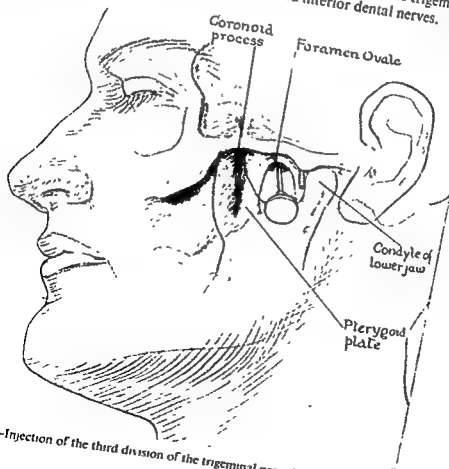


FIG. 138 — Injection of the third division of the trigeminal nerve by the lateral route.

*Relief
measures*

*Narcotics
and
analgesics*

Certain broad principles and instructions can be laid down. The whole manoeuvre of injection should be carefully planned and manipulations should be carried out gently and without hurry, care being taken not to damage neighbouring and important structures. The surest way of avoiding damage and of ensuring a satisfactory injection is to make the injection whilst the patient is conscious, so that he can give information regarding zones of developing

*Principles
of treatment*

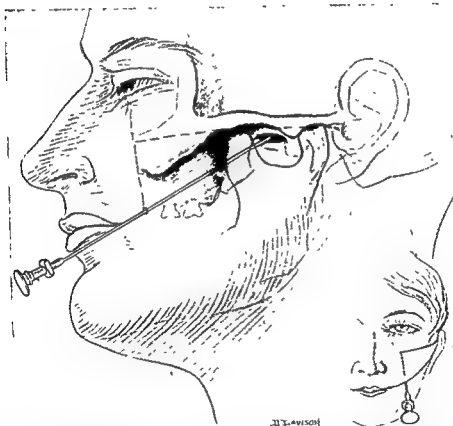
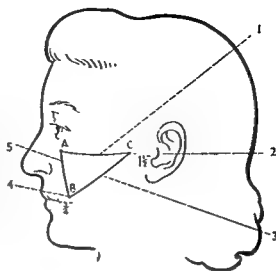


Fig. 139—Injection of the third division of the trigeminal nerve and of the Gasserian ganglion by the anterior route. The needle is introduced at the entrance point (B) and pushed backwards and upwards, aiming at the point (C), the needle is so angled that it runs in the planes (B)-(C) and (B)-(A). (1) Inferior zygomatic border; (2) external auditory meatus; (3) line which runs from angle of mouth to point $1\frac{1}{2}$ inches in front of external auditory meatus; (4) point of entrance of needle; (5) line which runs from point of entrance towards pupil.

anaesthesia. To allay anxiety, $1\frac{1}{2}$ –3 grains of Sodium Amytal may be given half an hour before the operation. Morphine, in my experience, is unsatisfactory as it makes the patient either too drowsy to co-operate or sick when disturbed. When, for any reason, a general anaesthetic is necessary, the needle should be introduced at what is thought to be the correct site. A skin-gram should then be taken to confirm the position of the end of the needle and finally the patient should be allowed to come round sufficiently to



Superficial landmarks

FIG. 140.—Points to note when injecting the infra-orbital nerve. It is important to protect the eyeball from injury by placing a finger along the inferior orbital margin. The needle is introduced at right angles to the face half an inch laterally from the naso-labial fold and half an inch upwards from the bottom of the nostril. The foramen itself is found by tapping and probing.

The Novocain test

topography of the head and face that he can visualize the deeper bony structures through the skin. Also, with practice, an operator is able to localize and recognize the main bony buttresses by tapping with the end of the needle. A few minims of 1 per cent procaine hydrochloride (Novocain) are injected with a hypodermic needle into the skin at the point indicated. The injection needle is then withdrawn.

When the needle is inserted, in fact, minims of Novocain are injected. If the rests between the nerve fibrils, some degree of anaesthesia will develop in the area concerned—certainly within 2 minutes. If the test is satisfactory, a few minutes are allowed for the Novocain to disperse so that it does not dilute the alcohol, and then the absolute alcohol is injected slowly, a minim at a time,

co-operate before the injection proper is started. Absolute alcohol is the best fluid to inject; no more than $1\frac{1}{2}$ cubic centimetres should be injected into the Gasserian ganglion itself, with a maximum of 1 cubic centimetre into a division and only 1–2 minims into the more peripheral branches. It is always dangerous to have more than the chosen amount of fluid in the syringe at any one time, as too much alcohol will be injected inadvertently into the tissues should the plunger slip. It is also wise to keep the coagulating fluid in a coloured receptacle so that it cannot be mistaken for Novocain.

When making deep injections superficial landmarks are an aid. My belief, however, is that such markings can be of only limited value. It is essential, if an operator is to become really skilled in this manoeuvre, for him to be so familiar with the topography of the head and face that he can visualize the deeper bony structures through the skin. Also, with practice, an operator is able to localize and recognize the main bony buttresses by tapping with the end of the needle. A few minims of 1 per cent procaine hydrochloride (Novocain) are injected with a hypodermic needle into the skin at the point indicated. The injection needle is then withdrawn.

until dense anaesthesia can be charted in the requisite area. It is important to leave the needle in position for at least 15 minutes so that further injections can be made should the anaesthesia show any sign of receding. It is unwise, when carrying out the Novocain test, to inject large quantities of Novocain, because perineural bathing can give rise to temporary anaesthesia; on the other hand, nothing but harm will come of alcohol injected into the perineural tissues.

(3) Operations

The various operative techniques are illustrated in Figs. 141, 142 and 143. Apart from intracranial neurectomies of the maxillary division at the foramen rotundum, other neurectomies are very rarely carried out and are of doubtful value. Personally, I often divide the second division intracranially at the foramen rotundum when the pain is strictly confined to the cheek, or when I am carrying out a fractional root section and am particularly anxious to leave the sensation of the cornea intact, but feel that a complete anaesthesia of the second division is essential.

*Value of
neurectomies*

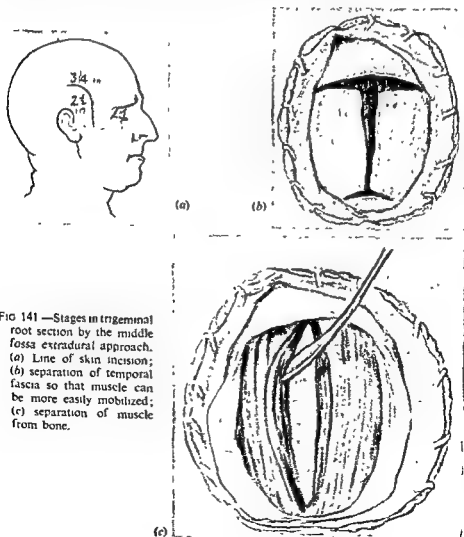
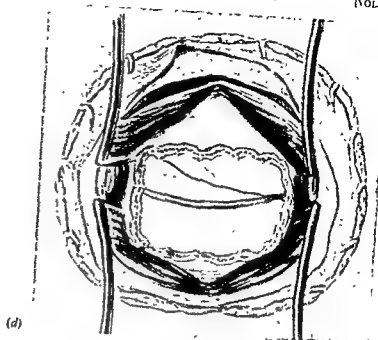
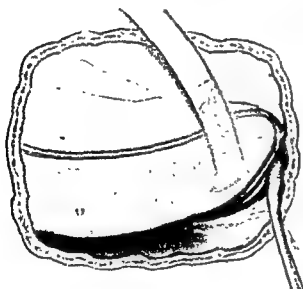


FIG 141 —Stages in trigeminal root section by the middle fossa extradural approach. (a) Line of skin incision; (b) separation of temporal fascia so that muscle can be more easily mobilized; (c) separation of muscle from bone.



(d)



(e)

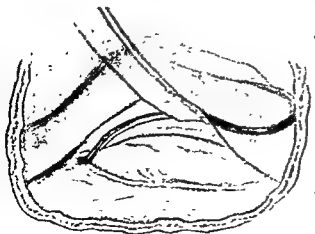


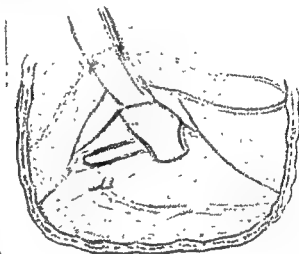
FIG. 141 (cont.)—
 Stages in trigeminal
 root section by the
 middle fossa extra-
 dural approach (d)
 Removal of bone—
 note the line of the
 middle meningeal
 artery, posterior
 branch; (e) and (f)
 lifting of dural en-
 velope from the base
 of the skull—note
 the bony mound (the
 centimetre barrier)
 which guards the
 third division of the
 trigeminal (the centi-
 metre barrier is a
 mound of bone at the
 base of the skull,
 which commonly
 stands up 1 centi-
 metre to the outside
 of the third division
 of the trigeminal,
 obscuring it and
 occasionally leading
 to technical diffi-
 culties in finding the
 root).



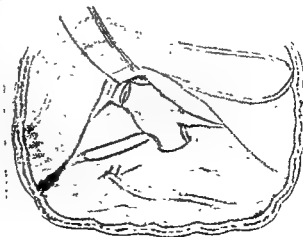
(g)

FIG. 141 (cont.).—

Stages in trigeminal root section by the middle fossa extradural approach. (g) Exposure of the third division at the foramen ovale, (h) exposure of the Gasserian ganglion, its upper part being enveloped in arachnoid mater and bathed by cerebrospinal fluid. The position of the great superficial petrosal nerve is clearly seen, (i) exposure of the posterior nerve root, the incision in the root shows fractional division of the mandibular and maxillary fibres.



(h)



(i)

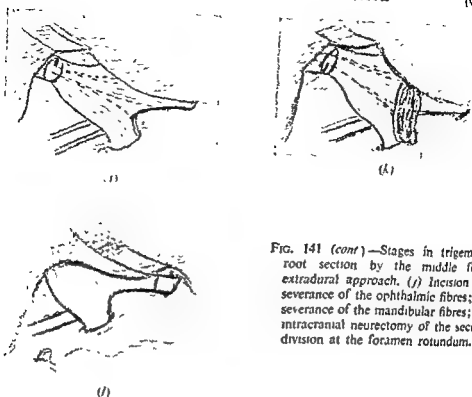


FIG. 141 (cont)—Stages in trigeminal root section by the middle fossa extradural approach. (j) Incision for severance of the ophthalmic fibres; (k) severance of the mandibular fibres; (l) intracranial neurectomy of the second division at the foramen rotundum.

7. ADVANTAGES AND DISADVANTAGES OF ROOT SECTION AND OF ALCOHOL INJECTION

(1) Root section

A limited denervation of the face is always possible. The motor nerve can be spared. Regeneration of the severed fibres of a posterior nerve root does not occur. The operation is one of great precision and certainty and, furthermore, root section can be carried out when, for technical reasons, injections have failed. The operation is carried out under general anaesthesia and no pain is inflicted on the patient; the post-operative course is smooth and painless.

A disadvantage is that a temporary facial paralysis may result.

(2) Alcohol injection

The main advantage of this procedure is that there is little danger to life and it can be carried out on those patients whose age or ill health preclude them from undergoing a major operative procedure. It is claimed that fractional injections of the ganglion are possible when great skill has been acquired. The third division can be injected when pain is restricted to this division, as it commonly is.

The disadvantages of injections, other than those into the ganglion, are that they do not give permanent relief; the pain is apt to recur within three years, and subsequent injections become more difficult because of fibrosis within the nerve sheath, which interferes with diffusion of the alcohol. My own inclination is strongly in favour of root section. In the hands of an expert the operation has become a relatively minor procedure. Broadly speaking, if a patient is strong enough to withstand a light general anaesthetic for three-quarters of an hour he is strong enough to undergo a root section.

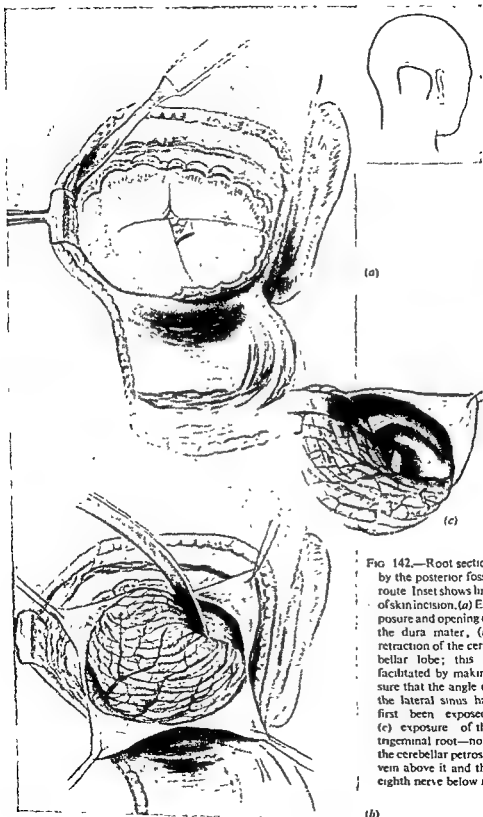


FIG. 142.—Root section by the posterior fossa route. Inset shows line of skin incision. (a) Exposure and opening of the dura mater, (b) retraction of the cerebellar lobe; this ■ facilitated by making sure that the angle of the lateral sinus has first been exposed; (c) exposure of the trigeminal root—note the cerebellar petrosal vein above it and the eighth nerve below it.

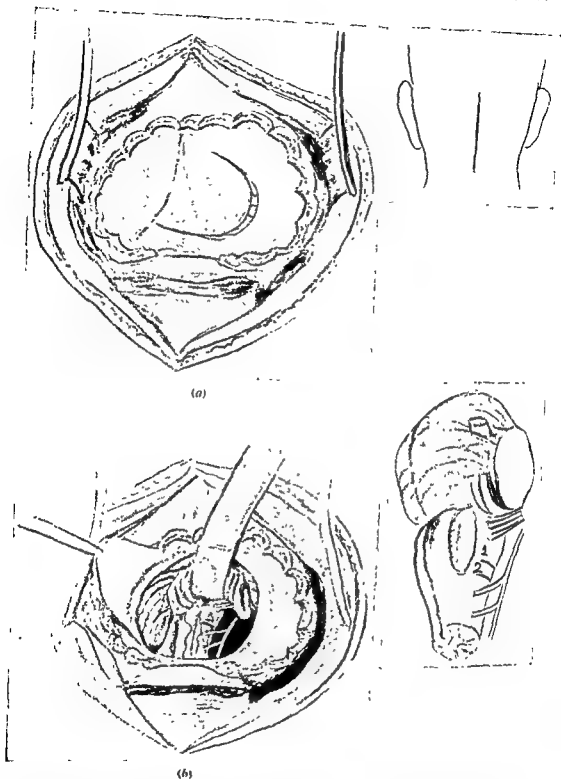


FIG. 143—The operation of trigeminal tractotomy. Insets show, respectively, midline skin incision and alternative levels for the incision into the tract. (a) Removal of bone and incision in the dura. (b) retraction of the cerebellar hemisphere upwards, showing the site of incision into the tract. The incision is 4 millimetres long and 3.5 millimetres deep at the levels shown in the tuberculum cinereum.

8. COMPLICATIONS FOLLOWING TREATMENT

(1) Keratitis

Keratitis is a trophic ulceration of the cornea resulting from denervation. At first the eye loses its gloss and the conjunctiva and cornea become steamy. Then a small ulcer appears on the cornea, sepsis soon intervenes, the eye becomes red, pus appears and the terminal result is perforation of the globe and destruction of the eye. In my experience, keratitis occurs only

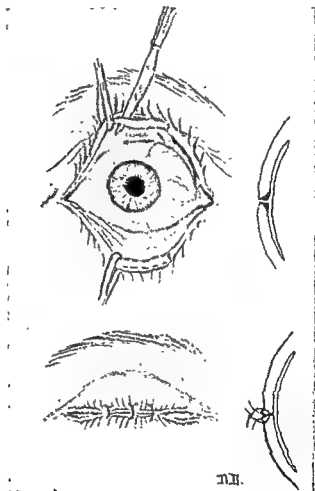


FIG. 144.—The operation of blepharorrhaphy for paralytic interstitial keratitis.

in those eyes in which, for some reason or other, lacrimal secretion ceases and the tears dry up. It is the insensitive dry eye that is prone to trophic complications.

Treatment

When severance of the ophthalmic fibres of the trigeminus is contemplated, *Prophylaxis* sterile liquid paraffin should be instilled into the conjunctival sac and the eye protected with a Buller's shield before operation. In no case should an attempt be made by the anaesthetist to elicit the corneal reflex

When the complication has once developed, the eye should be washed with saline, and penicillin drops instilled into the conjunctival sac, followed by drops of liquid paraffin. Also the eye should be protected with a Buller's shield. If, in spite of these simple measures, the condition does not resolve within 48 hours, the eyelids should be sutured together in the operation of blepharorrhaphy (Fig. 144). As an alternative to suturing the eyelids, a cervical sympathectomy may be performed. It has also been claimed that large doses of thyroid extract, given by mouth, will cure the condition.

Blepharorrhaphy or cervical sympathectomy

(2) Facial paralysis

Facial paralysis is an occasional sequel to root section and may develop immediately or after a few days. Usually the paralysis develops slowly and takes a few days to become complete. It results from traction on the great superficial petrosal nerve, and injury to the geniculate ganglion in the facial canal of the petrous bone. With electrical therapy this complication invariably resolves, although the patient may be left with a little over-action of the facial muscles and a wry smile.

Electrical therapy

(3) Trophic ulceration

Trophic ulcers occasionally develop in the mucous membrane of the nose and give rise to persistent nose bleeding. Trophic ulcers on the skin heal very rapidly with simple treatment.

9. RESULTS

The following statements are based on the figures of my own series of 500 cases of paroxysmal trigeminal neuralgia treated by one method or another. All patients whose faces had been rendered anaesthetic complained of a sense of numbness in the denervated area. The numbness was felt chiefly as a sensation of swelling and of coldness. However, 90 per cent of my patients were very well pleased with the results. The remaining 10 per cent complained of some degree of paraesthesia, of which a sense of itching behind the eye was the chief manifestation. Half of the patients in this latter group complained very little of their discomfort and it affected their behaviour negligibly; the other half stated that they were no better and some, in fact, avowed that they were worse.

Individual reactions

Some of the failures were due to mistakes in diagnosis and some to operating on patients who were later proved to be suffering from a frank psychoneurosis. Nevertheless, all told, the results were excellent, and in the whole realm of surgery one is not likely to meet with a more grateful group of patients than those who have been relieved of the pains of paroxysmal trigeminal neuralgia. Out of 200 cases which include all kinds of operation—trigeminal tractotomy, posterior root section and intracranial neurectomies—I have not had a single death either immediately or remotely that could be attributed to the operative procedure. In only one case was permanent damage done to the third nerve, and this was the result of carrying out a difficult dissection in the anterior end of the cavernous sinus. In only one case in 25 did a facial paralysis occur and in only one case was recovery from the paralysis unsatisfactory.

PART II

GLOSSOPHARYNGEAL NEURALGIA

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3. CLINICAL FEATURES — — — — —	267
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1. DEFINITION

A diagnosis of glossopharyngeal neuralgia implies that a patient is suffering periodically from sharp shooting pains in the area of distribution of the glossopharyngeal nerve. Moreover, these pains are not associated with objective sensory loss in the painful areas and are not due to any kind of pathological change that can be demonstrated either in life or at necropsy.

2. AETIOLOGY AND PATHOLOGY

The aetiology and pathology are unknown and the problem is similar to that of paroxysmal trigeminal neuralgia.

*Aetiology and
pathology
unknown*

3. CLINICAL FEATURES

The pain occurs in people beyond middle life. Typically, it starts in one side of the throat and shoots as a knife-like stab into the tonsillar region; occasionally it radiates deep into the ear. The pain is particularly brought on by swallowing food, but also saliva, the trigger-point being in the side of the throat or in the tonsillar fossa. There are intervals of freedom, but there is a tendency for the attacks to increase in severity and in frequency.

On examination, there is no objective sensory loss in the glossopharyngeal area, that is, in the tonsillar fossa, in the posterior third of the tongue and in the throat.

Regarding differential diagnosis, the problem is again similar to that in paroxysmal trigeminal neuralgia. However, great care must be taken to eliminate any possibility of the presence of carcinoma of the fauces, of the back of the tongue or of the tonsil.

4. TREATMENT

There is no known cure and, as in paroxysmal trigeminal neuralgia, the condition does not tend to burn itself out. Ease can be given by medical measures, but permanent relief demands operation. There are two types of operation, removal of the glossopharyngeal nerve in the neck, and removal of this nerve in the posterior fossa. Since recurrences are likely if the glossopharyngeal nerve is not severed above its branch to the ear, the intracranial operation is the one of choice (Figs. 145 and 146). The results of the operation are very satisfactory. It must be realized, however, that this is a ~~very~~ ^{Operation essential for permanent relief} serious condition indeed.

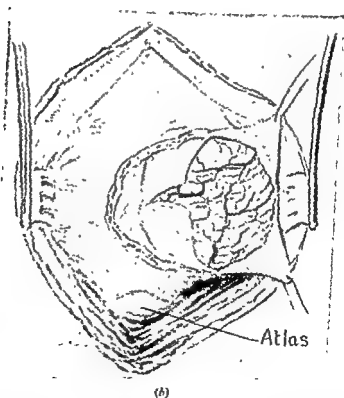
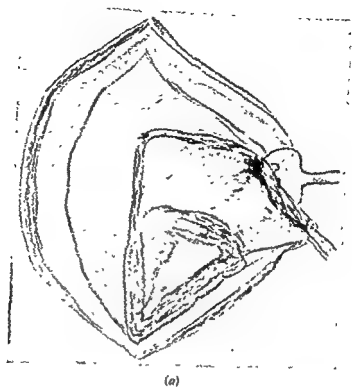
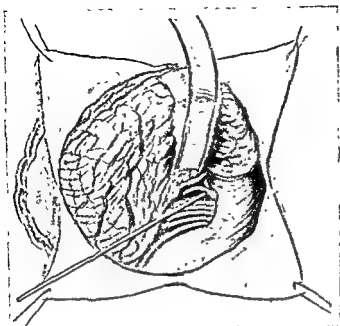
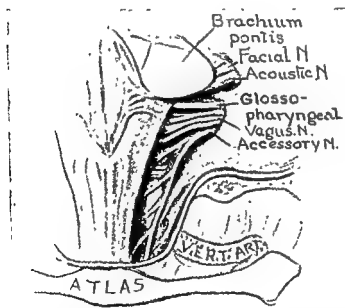


FIG. 145.—Stages in the operation for section of the glossopharyngeal nerve in the posterior fossa. (a) Reflection of the occipital muscles from the bone; (b) bone removal and opening of the dura mater.



(c)



(d)

FIG 145 (cont) —Stages in the operation for section of the glossopharyngeal nerve in the posterior fossa (c) Retraction of the cerebellar hemisphere and the identification of the glossopharyngeal nerve at the jugular foramen; (d) the anatomical relationship of the glossopharyngeal nerve.

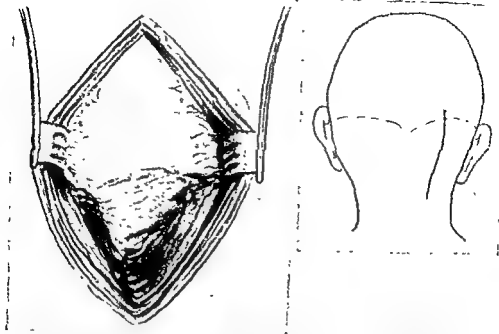


FIG. 146—Illustrating the alternative approach to that shown in Fig 145, for section of the glossopharyngeal nerve. Inset shows skin incision for lateral approach. The diagram shows separation of muscles and exposure of occipital bone.

BIBLIOGRAPHY AND REFERENCES

- Dandy, W. E. (1929) *Arch Surg., Chicago*, 18, 687.
 Fothergill, J. (1779). *Med. Observ. Inquiries*, 2nd ed., 5, 129.
 Gurlt, E., Wernich, A., and Hirsch, A. (1929) *Biographisches Lexicon*, 2nd ed. Vol. I, p. 133. Berlin and Vienna, Urban Scharzberg.
 Harris, W. (1926) *Neuritis and Neuralgia*. London; Oxford University Press.
 — (1937) *The Facial Neuralgias*. London; Oxford University Press.
 Hartel, F. (1912a). *Zbl. Chir.*, 39, 705.
 — (1912b). *Arch. klin. Chir.*, 100, 193.
 — (1929). *Ibid.*, 156, 374.
 Hartley, F. (1892). *N.Y. med. J.*, 55, 317.
 Horsley, V. (1891) *Brit. med. J.*, 2, 1139, 1191 and 1249.
 Jefferson, G. (1931). *Lancet*, 2, 397.
 Krause, F. (1892). *Arch. klin. Chir.*, 44, 821.
 — (1893) *Dtsch. med. Wschr.*, 19, 341.
 — (1896). *Die Neuralgie des Trigemini, nebst der Anatomie und Physiologie des Nerven*. Leipzig; Vogel.
 Lévy, F., and Baudouin, A. (1906). *Pr. méd.*, 14, 125.
 Mueller, G. (1875). *Die Prosopalgie und ihre Heilung durch die Neurectomie*. Inaugural dissertation. Kiel; Mohr.
 Sjoqvist, O. (1937). *Zbl. Neurochir.*, 2, 274.
 Spiller, W. G., and Frazier, C. H. (1901) *Univ. Pa. med. Bull.*, 14, 342.
 — (1933). *Arch. Neurol. Psychiat.*, Chicago, 29, 50.
 Sprengel, K. (1815–20). *Histoire de la médecine*. Trans. by Jourdan, A. J. L., Paris; Deterville.

[References to other titles are given under Neuralgia—Trigeminal, Glossopharyngeal in the Index Volume. The subject is also dealt with under the heading of Neuralgia, Glossopharyngeal and Trigeminal in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 174.]

NOSE, NASOPHARYNX AND ACCESSORY SINUSES

BY JOHN ANGELL JAMES, M.D., M.R.C.P., F.R.C.S.

SURGEON, EAR, NOSE AND THROAT DEPARTMENT, BRISTOL ROYAL HOSPITAL;
SURGEON, EAR, NOSE AND THROAT DEPARTMENT, BRISTOL CHILDREN'S
HOSPITAL

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PART I

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1. DEFORMITIES OF THE NASAL SEPTUM

244.] Deformities of the nasal septum may be traumatic or developmental. The deformity is of no importance unless the deviation obstructs the nasal airway or the ostia of the sinuses—so predisposing to sinusitis—or causes reflex neuralgia by pressure against the turbinates. The common complications are chronic rhinitis, sinusitis and infections of the pharyngo-tympanic (Eustachian) tube and lower respiratory tract.

(1) Differential diagnosis

Other causes of nasal obstruction which should be excluded are: (1) Alar collapse. This is relieved by abduction of the ala. (2) Nasal polypi, which are greyish in colour and mobile. (3) Enlarged turbinates, which are attached to the outer wall of the nose.

(2) Indications for surgical intervention

Submucous resection of the septum is performed when the obstruction is sufficient to cause persistent mouth-breathing or the secondary results of nasal obstruction described above.

(3) Operative technique

If the anterior end of the septal cartilage is dislocated the incision should *Incision* be made over the prominent anterior margin (Fig. 147 (a)), otherwise it

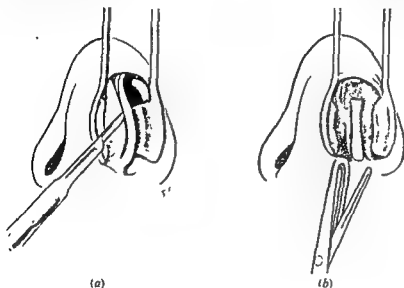


FIG 147—Submucous resection of the septum (a) incision, (b) second stage

should be made at the muco-cutaneous junction. The muco-perichondrium is elevated on both sides and the deflected area of bone and cartilage is removed with punch forceps (Fig. 147 (b)).

The results of this operation are excellent.

2. ACUTE RHINITIS

(1) Aetiology

The primary cause of the common cold is infection with a virus, and this is followed after a day or two by secondary infection with various species of *Infective* bacteria. The specific types include those due to diphtheria, syphilis and gonorrhoea. A similar acute infective rhinitis occurs in the specific fevers. The infection is carried by droplets, dust, baths or eating utensils. A primary aseptic rhinitis may be caused by local irritants or by the internal adminis- *Aseptic* tration of iodides.

(2) Pathology

The ciliated epithelium which forms the superficial layer of the nasal mucous *Ciliary* membrane is normally protected by a blanket of mucus, in constant movement *action* towards the nasopharynx. While this remains intact the underlying mucosa is protected. The factors which favour infection are the drying or washing away of the mucus, the lowering of general resistance, or special virulence or concentration of the infecting organism (Proetz, 1941)

(3) Clinical picture

Complications The symptoms, which are ushered in by a feeling of dryness and congestion in the nose, are too well known to warrant further description. The disease usually runs its course in from 5 to 10 days. The infection may spread to the accessory sinuses, pharyngo-tympanic tube or lower respiratory tract.

(4) Differential diagnosis

Vasomotor rhinitis The diagnosis lies between acute rhinitis and vasomotor rhinitis. Vasomotor rhinitis is periodic, and signs of infection are absent. The mucous membrane is purplish and the discharge watery.

(5) Treatment

Sprays In the early stages rest and a warm, even temperature are indicated, together with copious fluids, hot baths and the administration of salicylates. Steam inhalations are useful, and the airway should be maintained with isotonic vasoconstrictor. In the later stages the use, in addition, of penicillin, V, or penicillin

3. CHRONIC RHINITIS**(1) Definition and aetiology**

Types There are four types of chronic rhinitis: simple, hypertrophic, polypoid and atrophic.

Simple and hypertrophic rhinitis may be due to unhealthy atmospheric conditions, excessive use of tobacco or snuff, infected adenoids, nasal obstruction or chronic sinusitis.

The polypoid form develops when allergy and infection are combined

The atrophic form may be primary, or secondary to excessive removal of mucous membrane.

(2) Clinical picture

Simple and hypertrophic In the simple and hypertrophic forms the patient complains of nasal stuffiness, headache and discharge. The nasal mucosa is generally dull red in colour and swollen, particularly over the inferior turbinates, and does not respond to shrinkage (Appendices I and II).

Polypoid In polypoid rhinitis, polypi develop, and the signs and symptoms of both allergic and infective rhinitis are present

Atrophic In the atrophic form the nasal passages are excessively wide and the mucous membrane is thin, dry and covered with offensive greenish crusts (ozæna).

(3) Treatment

For the simple and hypertrophic forms local treatment is the same as that for acute rhinitis, and an alkaline douche (Appendix XI) may be used occasionally to remove thick discharge. Penicillin is useful in the form of a snuff, if the organisms are sensitive to it (Appendix VII).

Hormone treatment For atrophic rhinitis it is necessary to douche frequently. Oestradiol, 10,000 units in 1 cubic centimetre of oil of sesame, is used as a paint or spray to stimulate mucosal activity and proliferation. The offensive smell is reduced by painting the mucous membrane with 25 per cent glucose in glycerin (Fleming, 1946).

(4) Indications for surgical intervention

In hypertrophic rhinitis the turbinates may be reduced by linear galvano- *Partial*
cauterization or by very conservative excision of redundant mucous *turbinectomy*
membrane.

4. ALLERGIC RHINITIS AND SINUSITIS**(1) Definition and aetiology**

Allergic or vasomotor rhinitis and sinusitis may be caused by the following
allergens: physical agents, inhalants, drugs, ingestants and bacteria. *Allergens*

(2) Clinical picture

The predominant symptoms are attacks of sneezing, nasal obstruction,
irritation and watery discharge. The mucosa is swollen, purplish and hyper-
sensitive.

(3) Special aids to diagnosis

Nasal smears show excess of eosinophils. Skiagrams may show mucosal
thickening in the sinuses. The allergen may be identified by skin testing.

(4) Differential diagnosis

See under Acute Rhinitis.

(5) Treatment

Many methods of treatment are available, including: (a) avoiding the aller-
gen; (b) specific desensitization; (c) nasal ionization and (d) the use of the
following: calcium and vitamins C and D; Benadryl (anti-histamine action) *Benadryl*
in capsules of 50 milligrams, from 1 to 3 a day; vasoconstrictor vapours
(Appendix II), sprays and drops (Appendix I), which afford temporary
relief.

**5. INFECTIONS OF THE NASAL ACCESSORY SINUSES:
ACUTE****(1) Definition and aetiology**

Infection takes place by invasion, through the nose, by extension from a *Mode of*
dental focus or following injury. *infection*

(2) Clinical picture

The symptoms are those of an acute coryza, with headache, nasal discharge
and pain localized over the affected sinuses (Fig. 148). The pain is most
intense at midday. Tenderness may or may not be present over the affected
sinuses, and swelling and oedema of the superficial tissues may occur. Rhino-
scopic examination shows the changes typical of an acute rhinitis, with swell- *Rhinoscopic*
ing in particular of the middle turbinate, and a stream of pus may be seen *appearance*
issuing from the ostium of the sinus.

The complications include abscess formation, osteomyelitis of the cranial
bones (Fig. 149), cavernous sinus thrombosis, intracranial infections, and
spread of infection to the pharyngo-tympanic tube or lower respiratory
tract.

(3) Special aids to diagnosis

Transillumination of the antrum or frontal sinus is not always reli-
able. Skiagrams may show mucosal thickening (Fig. 150), fluid levels
(Fig. 151), generalized clouding or complete opacity (Figs. 152 and 153).

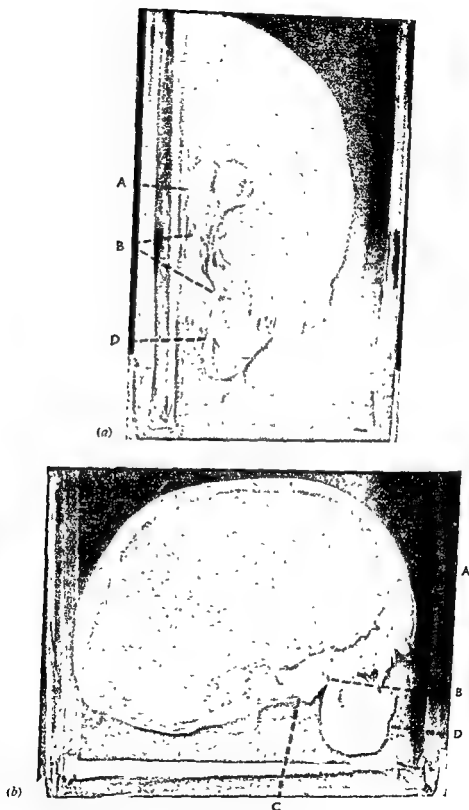


FIG 148.—Topography of accessory sinuses, etc (a) Lettering shows—A Frontal sinus B Ethmoid D Antrum (b) Semi-transparent half-skull. Accessory sinuses filled with metal. A Frontal sinus. B: Ethmoid. C. Sphenoid D Antrum.

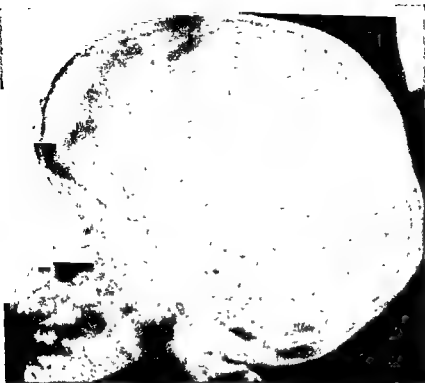


FIG 149 —Skigram of skull, showing osteomyelitis of the frontal bone.

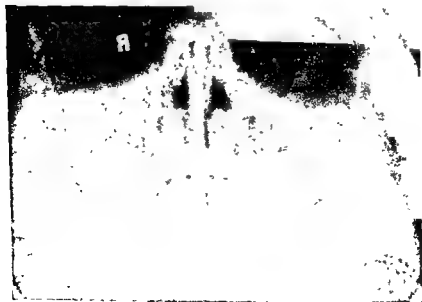


FIG 150 —Skigram in the vertex-mental projection, to show mucosal thickening in both antra.



FIG 151.—Skullgram in the vertico-mental projection: (a) to show fluid level in the right frontal sinus; (b) to show fluid levels in both antra.

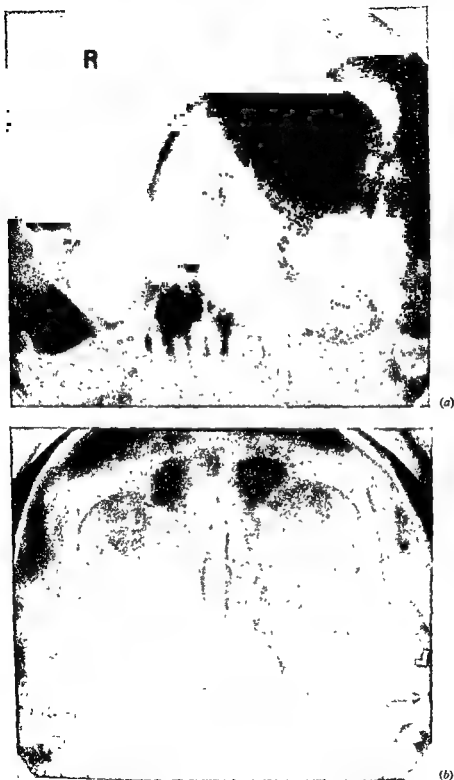


FIG. 152.—Skiagram in the vertico-mental projection: (a) to show opacity of the left antrum, ethmoid and frontal sinuses and obstruction of the nasal fossa with polyps; (b) to show opacity of the right antrum with dental root in the centre.

Bacteriological examination is important when prescribing treatment. Puncture and suction exploration may be employed in the later stages.



FIG 153—Skiagram in the occipito-frontal projection to show complete opacity of the ethmoid

(4) Differential diagnosis

Acute sinusitis may be simulated by the onset of acute specific fevers, rhinitis, infection of superficial tissues, angioneurotic oedema, dental abscesses or neuralgia. In these conditions other signs are present, the nose is normal, and if good skiagrams do not show any abnormality, sinusitis is improbable.

(5) Treatment

*Physiotherapy
and
chemotherapy*

The general and local treatments for acute rhinitis are supplemented for a week or more with daily ultra-short-wave irradiation, systemic penicillin and, if the condition is severe, with sulphonamides also.

If a sinus ostium is blocked, it may be released by applying a cocaine and adrenaline pack (Appendix VIII). Proetz displacement treatment is also of value when the ethmoid is infected. The object of the method is to introduce through the ostia small quantities of isotonic vasoconstrictor solutions, to which antiseptics or antibiotics may be added. The shrinkage of the mucous membrane around the sinus ostia improves the natural sinus drainage. Although the quantity introduced is small, there is usually sufficient to exert direct action on the sinus mucous membrane.

Technique

With the patient supine, and the head extended until the chin and external auditory meatus are in the same vertical plane, 2 millilitres of 0.5 per cent

ephedrine sulphate in normal saline solution (to which 2,000 units of penicillin per millilitre may be added) are instilled into each nasal chamber.

While the patient repeats "kick-kick-kick" to close the nasopharynx, negative pressure (180 millimetres of mercury) is applied for 1 second intermittently, at intervals of 1 second, through an olive tip fitted in one nostril, while the other nostril is closed with the forefinger.

Twelve alternations are usually sufficient to introduce the fluid into the sinuses (Proetz, 1946).

(6) Indications for surgical intervention

The majority of cases will resolve under conservative treatment. Surgery, however, will be required if conservative treatment fails, if complications develop or for the drainage of abscesses.

If the sinus ostium is blocked by a septal deviation or cystic middle turbinate, this should be corrected.

The next step should be puncture or catheterization. If puncture or catheterization fails, the sinus must be drained. *Sinus puncture*

If an orbital or superficial abscess is present over the antrum, frontal or ethmoidal sinuses, external drainage is indicated. If there is no abscess, the ethmoid or antrum may be drained intranasally. *External abscess*

(7) Operative technique

(a) Puncture and lavage

The antrum is punctured, using a trocar and cannula, beneath the inferior turbinate (Fig. 154). The posterior ethmoidal cells are punctured beneath the middle turbinate. The sphenoidal sinus is punctured through the anterior wall. The frontal sinus is catheterized through the natural ostium.

A specimen of the sinus contents, for examination, is obtained by suction, with a Watson-Williams syringe. If pus is present, the cavity is filled with a solution of penicillin in normal saline, 1,000 units per cubic centimetre or with proflavine and sulphonamide cream (Appendix XII). If the former is used, the cavity will require filling daily, either by repeated puncture or through an indwelling catheter, the cream will persist for from 5 to 7 days; puncture and lavage should then be repeated, and if pus is still present the sinus should be refilled. *Local chemotherapy*

(b) External drainage

(i) *Frontal and ethmoidal sinuses.*—The incision is made over the floor of the frontal sinus and lateral nasal wall (Fig. 155). The orbital or superficial abscess is drained, and a small opening may be made into the sinus, and a tube inserted for drainage.

(ii) *The antrum is drained by the Caldwell-Luc operation* (Fig. 158). (For description, see Infections of the Nasal Accessory Sinuses: Chronic.)

(c) Intranasal drainage

(i) *Ethmoid.*—The middle turbinate is amputated and the cell walls are removed with Stüder's punch forceps (Fig. 159).

(ii) *Antrum.*—The antral wall is opened beneath the inferior turbinate, with a harpoon or rasp, and the opening enlarged, avoiding the naso-lacrimal

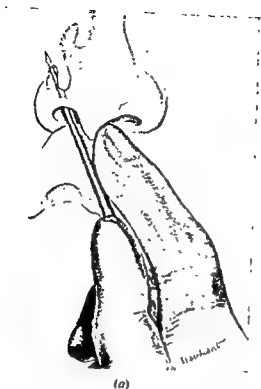
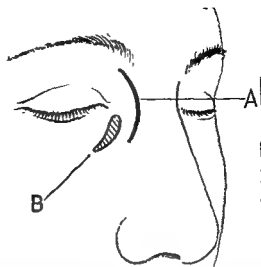


FIG. 154—(a) Antral puncture. Note forefinger against septum; (b) skiagram in the lateral projection, to show the antral cannula in position for lavage. The antrum has been filled with radio-opaque oil, which demonstrates the thickening of the mucous membrane.



FIG. 155.—External fronto-ethmo-sphenoidal operation
A: Incision. B, Lacrimal sac



duct and the posterior half-inch of the antral wall (Fig. 156) If the lower border of the inferior turbinate reaches the floor of the nose and obstructs the new ostium, a small portion should be cut off. *Inferior turbinectomy*

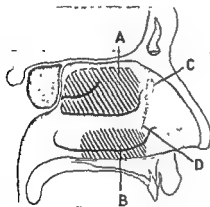


FIG 156 —Lateral wall of nose. A: Area opened during ethmoidectomy. B: Antral stoma after operation. C: Lacrimal sac. D: Naso-lacrimal duct.

(iii) *Sphenoidal sinus*.—Intranasal drainage is established by enlarging the ostium downwards to the floor.

6. INFECTIONS OF THE NASAL ACCESSORY SINUSES: CHRONIC

(I) Clinical picture

Types of infection

(i) *Latent types of infection*—In this form, the disease may only be discovered on making a routine x-ray examination for a focus of infection. The rhinoscopic examination may be entirely negative.

(ii) *Open*—The symptoms are nasal obstruction, discharge, cough and headache (Sluder, 1918). The headaches are usually intermittent, with

maximal intensity over the affected sinuses. The nasal mucous membrane is inflamed and the middle turbinate swollen. Discharge may be seen issuing from the sinus ostium.

(iii) *Polypoid*.—Polypi may develop in the sinuses and also in the nose.

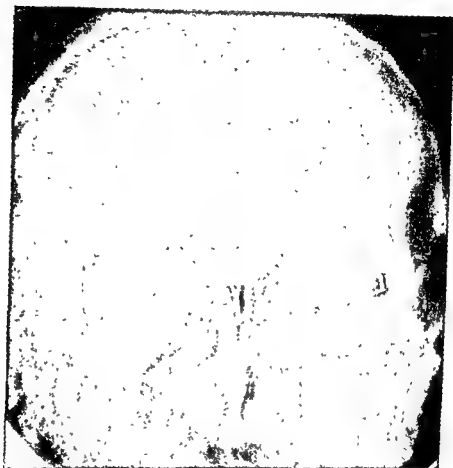


FIG. 157.—Skiagram in the occipito-frontal projection to show distension of the frontal sinus with mucocele, and destruction of the outer two-thirds of the supra-orbital margin.

(iv) *Mucocele*.—Gradual distension of the sinus causes displacement of its bony walls (Fig. 157).

(2) Complications

All those complications may occur which are described under Infections of the Nasal Accessory Sinuses. Acute, but the commonest are infection of the pharyngo-tympanic tube and lower respiratory tract, asthma, chronic toxæmia and osteitis.

(3) Special aids to diagnosis

For further details see Infections of the Nasal Accessory Sinuses: Acute. In doubtful cases mucosal swelling may be defined by filling the sinuses with radio-opaque oil (Fig. 154 (b)).

(4) Indications for surgical intervention

Mild cases may respond to conservative treatment. A number, however, will require some form of surgery.

A cystic middle turbinate, or deflected septum causing obstruction, should be corrected, and infected adenoids should be removed. *Removing obstruction*

If conservative treatment has failed, sinus puncture is indicated (as described under Infections of the Nasal Accessory Sinuses: Acute). If this fails, a drainage operation will be necessary, and this should be radical if the mucous membrane is very thick. *Choice of operation*

If polypi are present, a radical operation is indicated, unless the sinusitis is only of mild catarrhal type. In such cases the nasal polypi should be removed with a snare.

If the antrum is infected, a Caldwell-Luc operation is indicated; if the antrum and ethmoid are infected, a trans-antral ethmoid operation is preferred, and if a pan-sinusitis is present, the external fronto-ethmo-sphenoidal and antral operation should be performed.

An oro-antral fistula, if present, must be closed when the Caldwell-Luc antrostomy is performed. *Oro-antral fistula*

(5) Differential diagnosis

Confusion is most likely to occur when there is a chronic rhinitis and headache is the main symptom. X-ray examination is then of great importance. If the films do not show any abnormality, an active sinusitis is most improbable, but if any changes are seen, diagnostic puncture and lavage should be performed. In elderly patients malignant disease must be considered, and in such cases a biopsy should be performed. *Headache*

(6) Operative technique

(a) Caldwell-Luc antrostomy

A horizontal incision is made above the dental roots (Fig. 158) and the antrum is entered with gouge and mallet. If the mucosa is grossly thickened or polypoid, it is removed. A window is then cut into the nose beneath the inferior turbinate.

(b) Trans-antral ethmoidectomy

A Caldwell-Luc antrostomy is first performed, and the ethmoidal labyrinth is



Window into nose

FIG. 158 —Incision for Caldwell-Luc operation.

entered through the postero-superior area of the medial antral wall (Horgan, 1926) (Figs. 159 and 160).

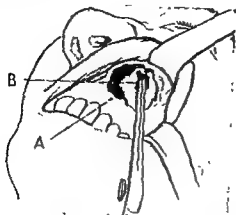


FIG. 159.—Trans-antral ethmoidectomy. A: Anterior wall of antrum. B: Margin of opening into ethmoid through medial antral wall.

FIG. 160.—Trans-antral ethmoidectomy, heavy shading represents area of ethmoid exenterated.

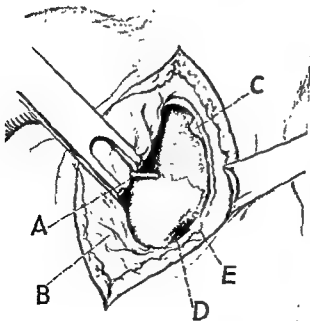
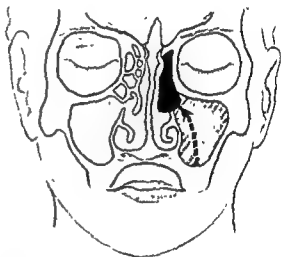


FIG 161.—External fronto-ethmo-sphenoidal operation A: Anterior ethmoidal artery. B: Orbital periosteum retracted. C: Attachment of pulley of superior oblique. D: Lacrimal fossa. E: Attachment of medial palpebral ligament.

(c) *External fronto-ethmo-sphenoidal operation* (Sewall, 1928)

(i) *Incision.*—An incision is made over the floor of the frontal sinus and lateral wall of the nose (Fig. 155).

(ii) *Elevation of the orbital periosteum.*—Great care is necessary to strip up the attachment of the pulley of the superior oblique muscle, the medial palpebral ligament and the lacrimal sac, with the periosteum (Fig. 161). Structures to preserve

(iii) *Ligation of the anterior ethmoidal vessels.*—These are found and ligated, or coagulated, as they enter the fronto-ethmoidal suture, at a depth of about 1 inch (Luongo, 1936) (Fig. 162). Ligation of ethmoidal arteries

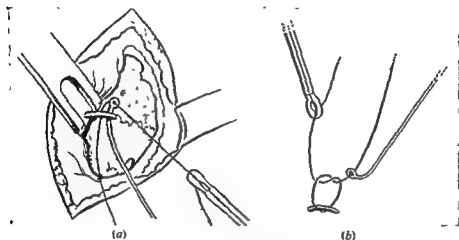


FIG. 162. (a) and (b).—External fronto-ethmo-sphenoidal operation. Ligation of anterior ethmoidal artery is shown

(iv) *Excision of bone*—The frontal sinus and ethmoid may then be entered, with gouge and chisel (Fig. 163). The whole of the floor of the frontal sinus is removed, and all ethmoidal cells are exenterated. Frontal and ethmoidal sinuses

(v) *Sphenoidal sinus*—The anterior wall of the sphenoidal sinus may then be opened.

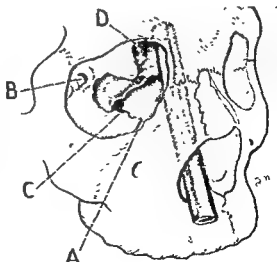


FIG. 163.—External fronto-ethmo-sphenoidal operation A. Lacrimal fossa. B. Optic foramen C. Cut edge of ethmoid D. Frontal sinus.

(7) Results of treatment

If the above-mentioned operations are carried out with the utmost care, and the inferior turbinate is preserved, the results are good.

7. TUBERCULOSIS OF THE NOSE**(1) Clinical picture**

Tuberculosis affects the anterior part of the septum and may take the form of a granuloma, ulcer or lupus vulgaris. The symptoms are nasal obstruction, bleeding, crusting and discharge.

Lupus

Lupus of the nose is often associated with lupus on the face and shows typical apple-jelly nodules.

(2) Special aids to diagnosis*Blanching*

Blanching with Liquor Adrenalinae defines the apple-jelly nodules. Bacteriological and histological examinations may be required.

(3) Differential diagnosis

Rhinitis sicca is recognized by the absence of infiltration around the ulcer margin.

Gummas usually develop in the posterior part of the septum.

Malignant tumours grow rapidly and bleed freely.

(4) Treatment**(a) Ulcers or granulomas**

The usual general measures, and alkaline douches with colloidal silver or oily sprays, are indicated (Appendices III, IV and XI).

(b) Lupus

Local application of chaulmoogra oil, and oral administration of 150,000 units of vitamin D daily (Dowling and Thomas, 1946) for 2 or 3 months are useful measures. If these fail, the lesion should be cauterized with acid nitrate of mercury, or by electro-cautery or diathermy.

8. NEOPLASMS OF THE NOSE: BENIGN

Excluding mucous polypi, which are not true tumours, the commonest tumours are papillomas and osteomas.

(1) Clinical picture*Osteomas
and
papillomas*

Osteomas interfere with the nasal airway or sinus drainage, or displace surrounding structures (Fig. 164). They throw a clean-cut shadow in the skiagram. Papillomas cause nasal obstruction and epistaxis.

(2) Differential diagnosis

An osteoma may resemble a rhinolith, but the latter is not firmly attached. The expansion of the sinuses, due to a mucocele or malignant neoplasm, may be distinguished by rhinoscopy, x-ray examination and biopsy.

(3) Indications for surgical intervention

Osteomas and papillomas are tumours which grow very slowly, but surgical removal offers the only cure.



FIG. 164 —Skiagram in the vertico-mental projection to show an osteoma of the ethmoid invading the orbit, nasal fossa and medial wall of the antrum

(4) Operative technique

Osteomas in the fronto-ethmoidal region are approached through a lateral rhinotomy or Caldwell-Luc incision unless there is a very massive intracranial extension, when they should be approached intracranially through a frontal flap. (For description of lateral rhinotomy, *see* p. 291.)

9. NEOPLASMS OF THE NOSE: MALIGNANT

(1) Morbid anatomy

Of malignant tumours of the nose and sinuses 60 per cent are squamous-cell epitheliomas (Ewing, 1940).

(2) Clinical picture

Pain is nearly always a prominent feature. Nasal obstruction, bleeding and discharge are usually present. The signs and symptoms depend on the site of origin.

*Invasion of
lymphatic
glands*

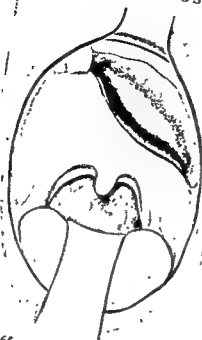


FIG. 165.—Carcinoma of antrum invading the hard palate.

demonstrate the neoplasm. When the invasion of surrounding tissues occurs the appearance and history may suggest sinusitis, dental abscess or tumour

(a) If in the antrum, there may be persistent pain in the teeth, followed by swelling of the palate or cheek (Fig. 165).

(b) If in the ethmoidal region, there is lacrimation, proptosis and displacement of the eye.

Secondary invasion of the cervical glands occurs in 30 per cent of cases, but distant metastases are rare.

(3) Special aids to diagnosis

A skiagram shows an opacity in the affected sinus, with displacement or destruction of the bony walls (Fig. 166).

(4) Differential diagnosis

In the early stages a diagnosis of neuralgia or sinusitis is commonly made, but x-ray examination rarely fails to

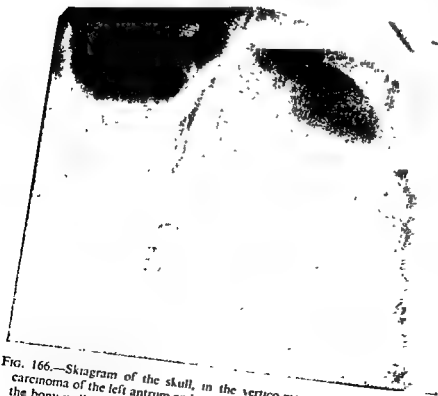


FIG. 166.—Skiagram of the skull, in the vertico-mental projection, to show carcinoma of the left antrum and ethmoid, with distension and destruction of the bony walls of these sinuses. The right antrum is also opaque.

of dental origin, but the skiagram shows destruction of bone, and a biopsy should always be performed.

(5) Indications for surgical intervention

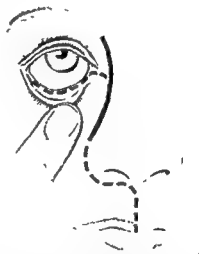
If the tumour is highly malignant or has invaded the base of the skull, x-ray irradiation is the best treatment. In other cases the mass should be excised by diathermy, and the cavity subsequently treated by x-ray irradiation and intracavitary gamma irradiation (Windeyer, 1943 and 1944).

(6) Operative technique

(a) The trans-palatal approach is most suitable for tumours involving the antrum. The incision encircles the hard palate, which is then removed, and diathermy is employed to excise the growth. The eye may have to be removed.

Bleeding is arrested by packing with gauze impregnated with proflavine and sulphonamide cream (Appendix XII); this may remain *in situ* for a week before it becomes offensive. It is rarely necessary to ligate the external carotid artery (Harmer, 1935).

(b) Lateral rhinotomy is indicated when the tumour is in the ethmoidal region. The incision in this operation begins beneath the inner end of the eyebrow and is carried downwards on the lateral wall of the nose (Moure, 1902) (Fig. 167).



Lateral rhinotomy

FIG 167.—Lateral rhinotomy. Primary incision (Dotted line indicates possible extensions.)

(7) Results of treatment

Fifty per cent of 5-year cures have been reported (New, 1938).

10. GENERAL MANAGEMENT OF THE PATIENT

(1) Pre-operative management

In addition to the usual general examination, any anaemia or tendency to undue bleeding should be investigated and treated, and vitamin C should be administered.

Sterilization of the area of operation is impossible, and if a chronic infection is present, or an extensive operation is to be undertaken, the administration of penicillin and sulphonamides should be commenced before operation.

(2) Anaesthesia

Minor operations are usually performed under local anaesthesia. For the remainder a combination of endotracheal inhalation anaesthesia and local anaesthesia, with light premedication, is preferred. Morphine must be used with care on account of its depressant effect on the cough reflex.

(i) *Surface anaesthesia* suffices for cauterization, sinus puncture or biopsy. For young children 2 per cent Decicain is less toxic than cocaine (Appendix X). For the remainder 5 per cent or 10 per cent cocaine solution may be used.

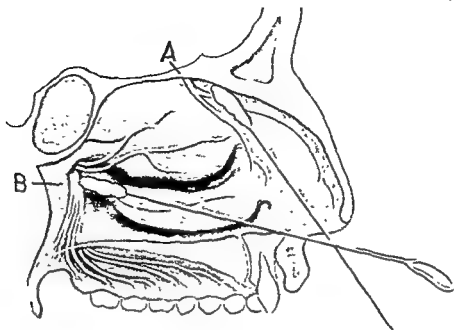


FIG. 168.—Trunk anaesthesia Nerve supply of lateral wall of nose; A: Anterior ethmoidal nerve, B: Spheno-palatine nerve.

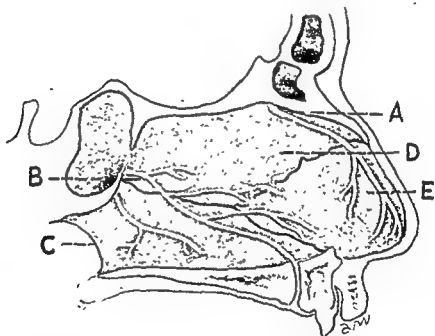


FIG. 169.—Nerve supply of nasal septum A: Anterior ethmoidal nerve B: Spheno-palatine nerve. C: Vomer. D: Perpendicular plate of ethmoid E: Septal cartilage.

as a spray or pack, or a trace of Ung. Cocainae (Appendix IX) may be smeared over the surface.

(ii) *Trunk anaesthesia* is obtained by the application of cocaine over the spheno-palatine nerve, behind the posterior end of the middle turbinate, and over the anterior ethmoidal nerve, above the middle turbinate (Figs. 168 and 169).

(iii) *Infiltration or block anaesthesia* may be produced by Novocain 1 per cent Premedica or 2 per cent or Nupercaine 0.1 per cent with Liquor Adrenalinae 1 : 100,000, and may be supplemented by premedication with phenobarbitone, grains 2, two hours before, and Omnopon, grain $\frac{1}{2}$, and scopolamine, grain $\frac{1}{32}$, one hour before operation.

(3) Haemorrhage

The blood supply of the nose (Fig. 170) is derived from the following sources:

(i) *Branches of the external carotid.*—These supply the middle and inferior meatus, the inferior turbinate, the antrum and the major portion of the nasal septum.

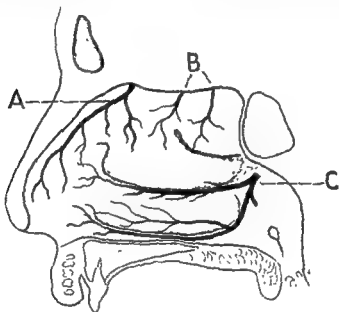


FIG. 170.—Arteries of the lateral wall of the nose A. Anterior ethmoidal artery B. Posterior ethmoidal artery. C: Sphenopalatine artery.

(ii) *Branches of the internal carotid.*—The anterior and posterior ethmoidal arteries supply the ethmoidal and frontal sinuses, and the lateral nasal wall and septum from the roof to the level of the lower border of the middle turbinate.

(a) Varieties of haemorrhage

Spontaneous haemorrhage may be due to varicosities of the vessels in Little's area of the septum, hyperpiesia, blood diseases, infections or tumours.

Traumatic haemorrhage may follow an operation, nasal injury or fracture of the skull.

(b) Treatment

When the bleeding point can be seen it is best to coagulate it with galvano- Cauterization cautery. If the bleeding point is not accessible sedatives should be given and

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1. ACUTE AND CHRONIC NASOPHARYNGITIS

(1) Aetiology

Acute, chronic and atrophic nasopharyngitis have a similar aetiology and pathology to the corresponding lesions in the nose.

(2) Clinical picture

(i) *Acute nasopharyngitis* is usually associated with acute rhinitis, and the *Symptoms* general signs and symptoms are the same. The local symptoms consist of burning and soreness, post-nasal discharge and obstruction. On posterior rhinoscopy the mucous membrane is seen to be red and swollen, with mucus *Rhinoscopy* or muco-pus on the surface.

(ii) *Chronic nasopharyngitis* rarely causes any general symptoms, but *Symptoms* occasionally there is some toxæmia. The local symptoms are those of post-nasal discharge and cough, and a feeling of stuffiness. On posterior rhinoscopy the mucous membrane is found to be inflamed, and the lymphoid *Posterior rhinoscopy* tissue and adenoids, if present, are swollen, with blobs of mucus or muco-pus on the surface.

(iii) *Atrophic nasopharyngitis* is usually associated with atrophic rhinitis and the clinical picture is similar.

The complications are: lymphadenitis, tubal infection, infection of the pharynx and lower respiratory tract, anorexia, dyspepsia and alimentary infection due to swallowing infected discharge.

(3) Special aids to diagnosis

Naso-endoscopy is useful if posterior rhinoscopy is impossible, and also for *Naso-endoscopy* investigating the tubal orifice. Lateral skiagrams of the nasopharynx will show the depth and extent of adenoids or other swellings.

Nasopharyngoscopy, with a Yankauer's nasopharyngoscope, and palpation with the finger, should be performed under anaesthesia. The former provides *Naso-pharyngoscopy* a direct view of the vault and fossae of Rosenmüller.

Bacteriological examination is used for guidance in treatment.

(4) Differential diagnosis

The examination should include a careful search for the primary cause in the *Primary causes* nose, accessory sinuses or tonsils.

Adenoids and choanal polypi are excluded by posterior rhinoscopy.

Gummas and tuberculous ulcers present typical characteristics.

Malignant tumours cause bleeding, obstruction and discharge if fungating, but the primary lesion is often very small. The glands are invaded at an early stage.

(5) Treatment

Acute and chronic forms are treated with steam inhalations, and sprays or drops introduced through the nose with the patient in the supine position, together with local and general chemotherapy, as used for acute rhinitis. Chronic forms, when due to hypertrophy of the adenoids, recover rapidly after removal of the adenoids. For the remainder, treatment is directed to removal of the primary cause, with local treatment as above.

2. ADENOIDS AND ADENOIDITIS

(1) Actiology

The important factors which cause chronic infection and hypertrophy of the adenoids are mouth-breathing, repeated colds or overwhelming infection during the infectious fevers.

(2) Clinical picture

(a) Obstructive type

The symptoms are mouth-breathing, snoring and nasal speech.

(b) Infective type

These patients are very susceptible to colds. There is persistent cough and post-nasal discharge. The complications are lymphadenitis, sinusitis, tubal and middle-ear infections, asthma and infections of the lower respiratory tract, anorexia due to swallowing infected discharge, and chronic toxæmia.

(3) Special aids to diagnosis

Lateral skiagrams of the nasopharynx are of value to show the size and depth of the adenoids.

The electric naso-endoscope, passed through the nose, is used when posterior rhinoscopy is impossible, and also for the accurate diagnosis of tubal or residual or recurrent adenoids, particularly in cases of deafness in children, and of barotrauma in airmen.

Palpation is usually performed only under anaesthesia.

(4) Diagnosis

The diagnosis presents little difficulty, provided that a reliable history is obtained and posterior rhinoscopy is tolerated by the patient.

The other causes of nasopharyngeal obstruction and infection are considered under Acute and Chronic Nasopharyngitis.

(5) Indications for surgical intervention

It is difficult to define the stage at which adenoid enlargement passes from the physiological to the pathological. The decision is reached on the history of obstruction and repeated infection, the rhinoscopic examination and the history or presence of complications. Small recurrent or residual adenoids, and the "tube tonsils" causing deafness, recurrent otitis or barotrauma may be treated by irradiation.

Complications

Naso-endoscopy

Irradiation

Radon or radium is inserted in a metal carrier, which is passed through the nose until it lies across the tubal elevation, where it is strapped in position. The dose delivered, at the surface of the applicator, is, according to Crowe *Dosage* and Burnam (1941), 640 r, repeated if necessary at intervals of 4 weeks.

(6) Operative technique

If the tonsils are to be removed at the same time, a Davis gag is used, with the patient in the Rose position.

A La Force adenotome may be used, but the shape does not suit every *La Force adenotome* nasopharynx. The most satisfactory instrument is a curette. The curette is passed over the adenoids; it is then held rigidly in the sagittal axis of the body, and the adenoids are swept off with a circular motion, following the contour of the nasopharyngeal roof and posterior wall (Fig. 171).

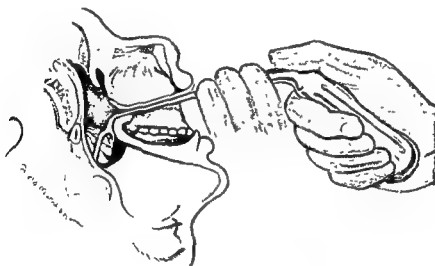


FIG. 171 — Removal of adenoids in Rose position.

Lateral extensions of the adenoid mass are removed by subsequent sweeps, always maintaining the curette in the same axis and cutting only on the roof and posterior wall. If adenoids alone are being removed, the operation may be performed with the patient in the lateral position.

(7) Results of treatment

Results are extremely satisfactory if the operation has been thoroughly performed and no adenoid tags have been missed. Nevertheless, in spite of every care, and particularly when there is chronic sinusitis or the adenoids have been removed in infancy, recurrences may take place with return of the *Recurrence* original signs and symptoms.

3. TUMOURS OF THE NASOPHARYNX: BENIGN

Benign tumours of the nasopharynx are rare, except choanal polypi, which *Choanal* are not true neoplasms. They include juvenile basal fibro-angioma, dermoids *polyp* and cysts, adenomas and neurofibromas.

(1) Clinical picture*Juvenile basal angioma*

The symptoms of obstruction, discharge and bleeding commence in childhood. Later, tubal obstruction and deafness develop, and this is followed by pain and invasion of the facial bones. At the age of 25 years the growth may atrophy.

The remaining tumours cause the symptoms and signs of nasopharyngeal obstruction.

(2) Indications for surgical intervention

Operative removal should be undertaken if the growth has not invaded the bones. If more extensive, it should be treated by x-ray irradiation or interstitial irradiation.

(3) Operative technique

The nasopharynx is exposed by splitting the soft palate in the midline, and it is usually necessary to remove some of the posterior margin of the hard palate with a rongeur. The tumour is then scraped off, through its attachment to the base of the skull.

4. TUMOURS OF THE NASOPHARYNX: MALIGNANT

These tumours represent about 2 per cent of all growths in the nose and throat; 80 per cent occur in men, although a number occur in childhood.

(1) Morbid anatomy

Of these tumours 70 per cent are carcinomas, mainly of transitional type. Characteristically, they tend to infiltrate deeply rather than to fungate. The infiltrating type follows the contour of the bones of the base of the skull and spreads into the foramina and forwards into the pterygoid fossa, orbit or nose. The lymphatic vessels are numerous and drain into the retropharyngeal, cervical and spinal accessory chains. Invasion occurs early in the course of the disease.

(2) Clinical picture**(a) Latent type**

A mass of secondary glands appears below the tip of the mastoid, and the primary lesion is discovered only on very careful examination.

(b) Primary nasopharyngeal type

The symptoms are nasopharyngeal bleeding, discharge and obstruction, with pain referred to the ear, and later obstruction of the pharyngo-tympanic tube and deafness. The growth is readily seen on posterior rhinoscopy.

(c) Cranial nerve type

Pain is usually an early symptom, followed by paralysis of the ninth, tenth, eleventh, twelfth and later the fifth and sixth cranial nerves.

(3) Special aids to diagnosis

X-ray examination will show soft-tissue swelling as well as bone destruction. Naso-endoscopy, nasopharyngoscopy and palpation under anaesthesia may be necessary, and biopsy should be performed.

Other
tumoursX-ray
irradiationLymphatic
drainage

Symptoms

(4) Indications for surgical intervention

Thorough surgical removal is impracticable and all cases should be treated by irradiation. The most successful method is to combine x-ray irradiation with intracavitary irradiation in divided doses. The radon or radium capsule is held in place by means of a rubber tube passed through one nostril and out of the other, or by special applicators. The lymphatic gland metastases are also treated by x-ray irradiation (Martin and Blady, 1940). *Technique*

(5) Results of treatment

The majority of these tumours are extremely radio-sensitive, and if treatment is begun at an early stage 20 per cent of 5-year cures may be obtained, and in the majority regression follows for a period of from 2 to 3 years.

PART III

APPENDICES: FORMULARY

APPENDIX I	Ephedrine or Paredrine	~	-	1%
	Saline Solution 0.9%	~	-	ad 3j
APPENDIX II	<i>Inhaler</i>			
	Benzedrine or Methedrine			
APPENDIX III	Argyrol	~	-	10%
	Ephedrine	~	-	1%
	Saline Solution 0.9%	~	-	ad 3j
APPENDIX IV	Eucalyptol	~	-	℥ ij
	Oil of Cinnamon	~	-	℥ ij
	Chloretone	~	-	℥ ij
	Liquid Paraffin	~	-	ad 3j
APPENDIX V	<i>Sulphonamide Spray or Drops</i>			
	Sulphanilamide or Sulphathiazole	~	-	1-5%
	Ephedrine or Paredrine	~	-	1%
	Saline Solution 0.9%	~	-	ad 3j
APPENDIX VI	<i>Penicillin Spray or Drops</i>			
	Penicillin	~	-	1,000 units
	Ephedrine	~	-	1%
	Saline Solution 0.9%	~	-	1 c.c.
APPENDIX VII	<i>Penicillin Snuff</i>			
	Powdered Penicillin	~	-	1,000 units
	Glucose	~	-	1 gramme

APPENDIX VIII

Cocaine and Adrenaline Spray, or Pack

Solution of Cocaine Hydrochloride 5-10%	9 parts
Solution of Adrenaline Hydrochloride (1:1,000)	1 part

APPENDIX IX

Ung. Cocainae

Cocainae Pur.	5ij
Thymol.	gr. j
Desicc. Suprarenal (1:5)	gr. xxiv
Paraff. Liq.	℥ 220
Paraff. Moll.	ad 3j

APPENDIX X

Solution of Decicain 2%	9 parts
Solution of Adrenaline Hydrochloride (1:1,000)	1 part

APPENDIX XI

Alkaline Nasal Douche

Sod. Bicarb.	3j
Sod. Bibor.	3j
Sod. Chlorid.	3ij
Menthol.	gr. iv

A teaspoonful in a tumblerful of warm water.

APPENDIX XII

Proflavine and Sulphonamide Cream

Sulphadiazine	1 part
Sulphanilamide	2 parts

In aqueous solution of isotonic buffered Proflavine 0.1% sufficient to make a thin cream.

APPENDIX XIII

Antrum Paste

Powdered Sulphanilamide	2 parts	} 20%
Powdered Sulphadiazine	1 part	
Lanette Cream	79 8%	

(16% Lanette Wax SX in water)

When required for use 15,000 units of Penicillin are dissolved in 3 or 4 c.c. of sterile water, and mixed with 14 c.c. of the paste.

REFERENCES

- Crowe, S. J., and Burnam, C. F. (1941) *Ann. Otol., etc., St Louis*, 50, 15.
 Dowling, G. B., and Thomas, E. W. P. (1946) *Brit. J. Derm.*, 58, 45.
 Ewing, J. (1940). *Neoplastic Diseases. A Treatise on Tumours*, 4th ed. Philadelphia; Saunders.
 Fleming, A. (1946). *Penicillin. Its Practical Application*. Ed. by Fleming, A. London; Butterworth.
 Harmer, W. D. (1935). *Lancet*, 1, 129.
 Horgan, J. B. (1926). *J. Laryng.*, 41, 510.

- Luongo, R. (1936). *Laryngoscope*, St. Louis, 46, 1.
- Martin, H. E., and Blady, J. V. (1940). *Arch. Otolaryng.*, Chicago, 32, 692.
- Moure, E. J. (1902). *Rev. hebdomadaire de Laryng.*, 22, 401.
- New, G. B. (1938). *Amer. J. Surg.*, 42, 170.
- Proetz, A. W. (1941). *Essays on the Applied Physiology of the Nose*. St. Louis; Annals Publishing Co.
- (1946). *The Displacement Method of Sinus Diagnosis and Treatment*, 3rd ed St. Louis, Zimmerman-Petty.
- Sewall, E. C. (1928). *Arch. Otolaryng.*, Chicago, 8, 144
- Sluder, G. (1918). *Concerning some Headaches and Eye Disorders of Nasal Origin*. St. Louis; Mosby.
- Windeyer, H. W. (1943) *Brit. J. Radiol.*, 16, 362.
- (1944). *Ibid.*, 17, 18

[References to other titles are given under Nose, Nasopharynx and Accessory Sinuses in the Index Volume The subject of Accessory Sinuses of the Nose is also dealt with in the *British Encyclopaedia of Medical Practice* (1936), Vol 1, p. 77.]

ODONTOMES AND EPITHELIAL CYSTS

BY PROFESSOR EVELYN SPRAWSON,
M.C., D.Sc., F.D.S., M.R.C.S., L.R.C.P.

CONSULTING DENTAL SURGEON, LONDON HOSPITAL; FORMERLY PROFESSOR
OF DENTAL SURGERY, UNIVERSITY OF LONDON; LECTURER ON DENTAL
SURGERY, LONDON HOSPITAL

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1. DEFINITION

245.] The true odontomes consist of: complex odontomes; compound odontomes (really multiples of complex odontomes); dilated odontomes; fibrous "odontomes"; cementomes; and such epithelial odontomes (multilocular cysts) as are of dental origin; these last are dealt with in Part II under the heading "Epithelial Cysts of the Jaws".

2. AETIOLOGY

Definition

Odontomes are new growths having origin from dental formative organs.

(i) *Complex odontomes*.—The typical complex odontomes are believed to originate as do the multilocular cysts of dental origin (basal-cell carcinomas), but, originating from adult ameloblasts, they stimulate calcification and so ultimately check and limit their own growth.

(ii) *Compound odontomes*.—These are mainly of a similar nature, but may consist of from a few to several hundred small masses of calcified dental tissue; they are often accompanied by cyst formation, probably originating as many cysts of eruption, some of which coalesce.

(iii) *Dilated odontomes*.—These probably originate from an adenomatous growth from adult ameloblasts. They nearly always erupt, though in some

cases they do so incompletely; sometimes they are small and erupt in the normal arch, particularly in the region of the maxillary second incisor.

(iv) *Fibrous "odontomes"*.—These are said to occur in rickety subjects only. They are very rare and it is doubtful whether they should be included among the odontomes. The fibrous portion consists of a much-thickened tooth sac and encloses a tooth.

(v) *Cementomes*.—These are masses, sometimes extensive, of hyperplastic cementum, and as they may be vascular in some respects they resemble bone.

3. MORBID ANATOMY

Calcified odontomes (except cementomes) ultimately limit and stop their growth by calcification. The effects of trauma on a developing tooth may simulate a true odontome, so that a correct diagnosis cannot be made without an accurate history.

4. CLINICAL PICTURE

(i) *Complex, compound and dilated odontomes*.—Complex, compound and most dilated odontomes give rise to swelling; they tend to erupt and sometimes succeed, though the attempt at eruption may be incomplete and accompanied by infection and pain, the intensity of which may be due to pressure on neighbouring parts as well as to the infection. One tooth, or possibly more than one, is usually absent from the normal series.

(ii) *Fibrous "odontomes"*.—These give rise to swelling, and one tooth or more is absent from the normal series.

(iii) *Cementomes*.—These may give rise to no signs or symptoms except those occasioned by increase in size, pressure on neighbouring parts and infection; cemental hyperplasia of inflammatory origin also occurs.

5 SPECIAL AIDS TO DIAGNOSIS

X-ray films, in both vertical and horizontal planes, are a necessity, not only *Radiography* for diagnosis but also to indicate the position, direction and extent of the mass; also, in the case of compound odontomes, to indicate the number of calcified masses which are present, and the presence of unerupted or supernumerary teeth.

6 DIFFERENTIAL DIAGNOSIS

When odontomes are exposed by partial eruption the sense of touch with a probe is characteristic, and so is the density of the x-ray shadow of calcified dental tissue. They also have to be differentiated from unerupted and supernumerary teeth; these can be recognized by their shape, size and position when seen in skiagrams taken from different view-points.

7. PROGNOSIS

Odontomes, having once given rise to signs and symptoms, will all continue to do so if untreated, and in fact the symptoms will tend to increase in intensity.

8. INDICATIONS FOR TREATMENT

In view of the prognosis, odontomes should all be treated as soon after diagnosis as is convenient; in addition, some of them call for treatment because they are unsightly.

9. TREATMENT

Removal

All odontomes should be removed.

(1) Complex odontomes

These may be exposed already, but if not the imprisoning bone must be cut away, so that the odontome may be loosened in the direction of least resistance, by means of elevators, till it can be removed. If infected, the wound must be packed to arrest haemorrhage, the packing being removed as soon as possible and the wound kept free from debris by frequent lavage; if there is no infection the wound may be closed immediately. If it is judged that there is any risk of fracturing the mandible, splints should be prepared beforehand.

(2) Compound odontomes

These odontomes, often being accompanied by cyst formation, should be opened and the denticles or masses of dental tissue removed together with the whole of the containing cystic walls. If the odontome is a small and uninfected one the wound may be closed immediately, but if infected it must be packed and kept clean of debris till healing has taken place from the base.

(3) Dilated odontomes

When small these are treated like teeth for extraction, but if large they may require the removal of a certain amount of bone and the careful use of elevators to remove them completely.

(4) Fibrous "odontomes"

Because the thickened tooth sac has prevented eruption, this is a clean operation, and after removal of the tooth in its sac the wound may be closed immediately.

(5) Cementomes

After an initial incision over the mass, sufficient of the imprisoning bone to allow of the loosening of the mass should be cut away, so that the cementome may be freed by the use of dental forceps and elevators. If there is no infection the wound may be closed immediately; otherwise it should be packed and allowed to heal by granulation.

10. RESULTS OF TREATMENT

Resolution is usually complete, and if there has been no external wound there is no external scarring or deformity. Any disability from loss of tissue or teeth can readily be made good by a denture.

Complete resolution

PART II

EPITHELIAL CYSTS OF THE JAWS

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1. DEFINITION

The epithelial cysts of the jaws consist of: cysts of eruption, dental cysts; dentigerous cysts ("follicular odontomes"); multilocular cysts ("adamantinomas", "epithelial odontomes"); naso-palatine cysts.

2. AETIOLOGY

Cysts of eruption and dental and dentigerous cysts invariably have origin from the epithelial dental-formative organs. The stimulus to their formation is either the presence of a tooth the eruption of which is delayed, or the action of toxins around the apices of pulpless and infected teeth.

The multilocular cyst is a neoplasm akin to a basal-cell carcinoma.

The naso-palatine cyst is an inclusion cyst or dermoid

3. MORBID ANATOMY

All dental cysts and about 85 per cent of dentigerous cysts (the remaining 15 per cent being mainly those which involve third molars) are caused by the passage of micro-organisms and their toxins through the apices of teeth with dead and infected pulps. The toxins stir into activity the epithelial cell rests of Malassez, which are the remains of the epithelial sheath of Hertwig and are found in the periodontal membrane attaching a tooth to the alveolar bone. *Micro-organisms*

All cysts of eruption and nearly all dentigerous cysts involving third molars are due to the stimulation of these same epithelial cells, or those of the tooth band, by the delayed eruption of the tooth. Sometimes the delay is due to misplacement. *Delayed eruption*

4. CLINICAL PICTURE

All these epithelial cysts are usually first brought to notice by a swelling which is painless unless infected or injured.

(1) Cyst of eruption

The patient is commonly a child, with a small bluish fluctuant swelling overlying a tooth the eruption of which is overdue; those cysts, however, which involve third molars do not usually give any evidence of their presence till they have become dentigerous cysts. If injured by contact with an opposing tooth, cysts of eruption may be painful.

(2) Dental cyst

*Creptant
fluctuation*

This cyst may occur at any age in a person having a pulpless or infected tooth or stump, and is a hard swelling which is painless unless it is infected. The swelling usually shows through the thinner outer alveolar plate, though in the maxillary premolar and molar regions the swelling may present on the palate. As the cyst gets larger the bone becomes thinned out till the typical crepitant fluctuation becomes evident. Dental cysts arise mostly in connexion with the permanent teeth; occasionally the cyst may remain intact after the removal of the tooth which has caused it. Sometimes they arise from deciduous teeth, but as these are present for so short a time they are not so often seen.

(3) Dentigerous cyst

This usually arises primarily as a dental cyst in connexion with a deciduous tooth. Because the cyst cannot absorb the underlying permanent tooth it grows around and encloses it, so producing a dentigerous cyst. If the causative deciduous tooth has been lost the successional permanent tooth will be absent on inspection. Except when the third molars are involved, the patient is rarely over 15 years of age.

The dentigerous cyst involving a third molar usually arises primarily as a cyst of eruption, which similarly grows and surrounds first the crown and ultimately the whole of this late and slowly erupting tooth. These cysts are noticed later in life, sometimes not till the sixth or seventh decade.

(4) Multilocular cyst

*Sites of
origin*

*Basal-cell
carcinomas*

Although it may occur in any part of the jaws this cyst is most common at the angle of the mandible, at or behind the position of the third molar. The complete series of molar teeth may be present. Probably only a small percentage of them are of dental (tooth-germ) origin, and the majority—those at the angle of the mandible—are basal-cell carcinomas originating from the epithelium covering the mandible in that region. They are usually first noticed at about the age of 30–35 years.

Those in other parts of the jaws are sometimes of dental origin and give rise to signs and symptoms during the tooth-forming and erupting period; in these cases the causative tooth will be absent on inspection.

(5) Naso-palatine cyst

This cyst often fails to give rise to signs or symptoms throughout life, and is discovered by accident when the teeth are being examined radiographically for some other reason. They occur most frequently in the maxillary incisor

region at the lines of developmental union of embryonic processes. Irritation from the wearing of a denture often gives rise to the first sign or symptom of their presence, though abrasion from food may also do so. They usually present as an irritable swelling just behind the maxillary incisor teeth, but sometimes on the labial aspect of the alveolus in that region. If infected they may resemble alveolar abscesses.

5. SPECIAL AIDS TO DIAGNOSIS

(i) *Cyst of eruption*.—A skiagram will confirm the diagnosis but is usually unnecessary.

(ii) *Dental cyst*.—Should there be several teeth with infected pulp canals, skiagrams are essential to indicate which particular stump is the causative factor, and also to indicate the size and extent of the cyst, and whether it has burrowed along the cancellous portion of the jaw or encroached on the antral cavity.

(iii) *Dentigerous cyst*.—Skiagrams are a necessity for confirmatory diagnosis, and also to indicate the size and extent of the cyst, particularly in the case of third molars.

(iv) *Multilocular cyst*.—Skiagrams are necessary to show the extent of the growth and whether or not any teeth are involved in it, and to differentiate it from a dentigerous cyst involving a third molar. This growth is peculiar in that it usually extends almost to the tip of the coronoid process.

(v) *Naso-palatine cyst*.—Skiagrams from different view-points are an absolute necessity in order to differentiate these cysts from the rarefactions so commonly seen at the apices of pulpless or infected maxillary incisor teeth.

6. DIFFERENTIAL DIAGNOSIS

(i) *Dental and dentigerous cysts*.—The chief point in the differential diagnosis of dental and dentigerous cysts is the tooth which is present within the latter cyst, and is absent from the normal series on inspection of the mouth, though, rarely, it may be a supernumerary tooth. The age of the patient is another deciding factor. *Presence of tooth*

(ii) *Multilocular cyst*.—This is most commonly confused with osteoclastomas, and possibly also with lipoid granulomatosis, myxosarcoma, osteitis fibrosa diffusa or circumscripta, and other rare neoplasms. Removal of a piece of the cyst for biopsy, or the modified aspiration biopsy, can be an invaluable aid to diagnosis. Estimation of the blood calcium may be necessary to exclude osteitis fibrosa diffusa. With lipoid granulomatosis other manifestations of the Schüller-Christian syndrome may be present. *Biopsy*

7. PROGNOSIS

(i) *Cyst of eruption*.—If left alone it may rupture and bring about spontaneous cure, but it may also divert the normal course of eruption of the tooth it overlies and so result in its malposition.

(ii) *Dental and dentigerous cysts*.—If left alone these may become opened by a process of denudation of the overlying tissues and so become infected and

simulate chronic abscesses. Rarely, dentigerous cysts, especially when multiple, may cause serious facial deformity, as seen in the "pig-faced" people of travelling exhibitions of monstrosities in the past.

(iii) *Multilocular cyst*.—If left alone this cyst may extend and invade soft tissues as far down as the clavicle, or may invade the face and simulate the late stages of extensive rodent ulcer. The growth is almost always single, but metastases have been known to occur in a few cases.

(iv) *Naso-palatine cyst*.—If left alone the cyst may rupture or become opened by denudation, and if infected it will simulate a chronic alveolar abscess. On the other hand, if all causes of inflammation are removed early, the cyst often resolves to its earlier quiescent state.

8. INDICATIONS FOR TREATMENT

Except in the early stage of the naso-palatine cyst, the presence of any of the other epithelial cysts of the jaws is an indication that surgical treatment is necessary.

9. TREATMENT

(1) Cyst of eruption

If the cyst is small and not interfering with the eruption of the underlying tooth, simple puncture to allow the contents to escape will suffice, but if it is large enough to interfere with the eruption of the tooth the tissue overlying it should be removed.

(2) Dental cyst

Preliminary treatment

As a preliminary the field of operation should be cleansed by removing salivary deposits on the teeth and extracting all infected teeth except the one in immediate connexion with the cyst. When these wounds have healed the causative tooth should be extracted, and the outer wall of the cavity is then removed widely enough to ensure that the cyst cavity becomes part of the mouth cavity. It is best to remove the whole of the epithelial lining, because occasionally neoplastic growth has occurred in what was left.

Removal of epithelial lining

When a maxillary cyst has invaded the antrum it may be advisable to leave that part of the epithelial lining adjacent to the antral lining, or a permanent antral-oral opening may result. In this case the oral opening must be kept open so that, as healing takes place from the base, the cyst lining may be brought to the surface and its epithelium ultimately may blend with the oral epithelium. Except in these cases packing is necessary only to arrest haemorrhage and is best removed as soon as possible. Frequent lavage is desirable so as to keep the cavity clean.

Treatment of small cysts

Small cysts may sometimes be opened by a linear incision; the lining is removed and the wound immediately closed by a stitch.

(3) Dentigerous cyst

Treatment is similar to that of dental cysts, but in addition the tooth in the cyst must be removed. Occasionally, if the operation is done near the time when the tooth in the cyst should erupt and it is in good position, it may be left to erupt, for it will do so when the obstruction is removed.

(4) Multilocular cyst

To prevent recurrence, this cyst must be completely removed, and when it extends up the coronoid process this becomes extremely difficult. Partial removal, conserving the continuity of the mandible, is often undertaken, because the growth is usually slow, but ultimately resection of the part of the mandible involved has to be performed, and in these cases splints or stainless-steel wire ligatures (of not less than 0.4 millimetre in thickness) should be used to keep the parts in their natural position till a bone-graft operation can be done or suitable dentures fitted. Repeated incomplete removals have been known to conserve the continuity of the mandible for over 30 years before a resection had ultimately to be carried out. Neither x-ray nor radium therapy has been found to be of much value.

(5) Naso-palatine cyst

Where it has approached the surface, this cyst should be opened by a linear incision, and the lining should be removed; the cyst usually shells out readily but is sometimes attached to the naso-palatine canal above or to the palatine mucosa below.

10. RESULTS OF TREATMENT

A perfect result without external deformity may usually be expected in all except the multilocular cysts, in which the extent and the completeness of removal will govern the result.

BIBLIOGRAPHY

- Colyer, J. F., and Sprawson, E. (1942). *Dental Surgery and Pathology*, 8th ed. London; Longmans, Green.
 McGregor, L. (1935) *Acta Radiol., Stockh.*, 16, 254.
 Sprawson, E., and Keizer, W. R. (1933). *Dent. Rec.*, 53, 369.
 [References to other titles are given under Odontomes and Epithelial Cysts in the Index Volume.]

OEDEMA—TRAUMATIC

BY JOHN N. BARRON, F.R.C.S.ED.
SURGEON-IN-CHARGE, ROOKSDOWN HOUSE, PLASTIC SURGERY HOSPITAL,
BASINGSTOKE

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1. INTRODUCTION

246.] Post-traumatic oedema is an expression of the disturbance of tissue metabolism in response to injury. Clinically, it is of great importance because of its secondary effects upon tissues undamaged by the original injury, and its sequelae are often out of all proportion to those of the injury itself. The condition is largely preventable and when it occurs it can be alleviated by adequate treatment; little is yet known, however, about its cause or its progression.

2. CLINICAL FEATURES

It is, of course, understood that oedema will follow injury applied to any part of the body. In the head, neck and trunk, except in the case of severe burns of these regions, it is of little permanent significance as its dispersal is automatic and rapid, and its effect on function transient.

In the limbs, however, where many factors militate against fluid drainage, a chain of reactions which create lasting and disturbing disabilities may be set up by its presence.

The condition is seldom met with in the first two decades, but becomes increasingly frequent in subsequent age groups. It generally bears some relationship to the severity of the injury, although at times—as if the patient were exceptionally susceptible—it occurs after trivial trauma. It is most frequently seen in injuries below the knee or the elbow, and is to be expected after crush injuries, fractures, infections, burns, operations and sutured lacerations.

(1) Stage of oedema

Following one of these injuries, the local oedematous response spreads rapidly—in a matter of a few hours—to all parts of the limb distal to the site of injury. The swelling continues until the skin becomes tense, the wrinkles and creases disappear and movement at the joints is greatly reduced.

Secondary
effects on
undamaged
tissue

Importance
in the limbs

Age incidence

Common sites

The oedematous tissue pits on pressure in the early stages, but later this sign cannot be elicited. Subjectively the limb feels leaden and useless, and the raised tension produces considerable discomfort, which at times is described as constituting an intolerable and constant ache.

(2) Stage of sclerosis

During the second week, in the untreated case, the swelling subsides a little and becomes firmer in consistency. It is usually at this time that the patient makes a determined effort to move the joints and finds that this movement is painful. Pain may now become the predominant factor, both as a symptom and as a barrier to function. Its effect is to increase the immobility of the limb; this, in turn, is followed by the stage of atrophy.

(3) Stage of atrophy

The clinical picture now changes. The soft pitting swelling becomes harder and more brawny, as if a mesh-work of contracting tissue were squeezing the fluid out of the limb. The skin becomes thin and shiny, scaly on the extensor aspects and moist and soft on the flexor surfaces. In the hand, the fingers taper, the nails become thin and serrated and the eponychium adhering to the nail surface grows out on to the nail itself. Progressive fixation occurs in the joints which become relatively bulbous as the swelling subsides and, although simple flexion-extension movements may be present to some degree, the fine co-ordinated functions in the hand, originating in the intrinsic muscles, are lost.

(4) Circulatory disturbances

Circulatory disturbances are constantly found. The extremity is cold and cyanotic and temperature responses suggest that there is a spasm of the arterioles with a dilated and paralysed capillary bed. In cold weather this is particularly noticeable, and the lowered temperature in the limb further embarrasses whatever function may be present. *Arteriolar spasm*

(5) Bone changes

X-ray examination shows a progressive decalcification of the bones of the affected area. This change commences early and the rate of calcium absorption is greater than could be accounted for by disuse. *Progressive decalcification*

(6) Appearance at operation

At operation the three stages of the disease are easily recognizable. One is struck in the early case by the amount of stagnant fluid present in all the tissues. Muscles are swollen and tense, tendon sheaths are thickened and contain abnormal amounts of fluid, and joint capsules are softened and bulging. Presently, as the swelling subsides, the picture becomes one of adhesion. Tissue planes which normally separate easily have to be stripped or cut. Fibrin surrounds the moving structures and the neuro-vascular bundles. Later, there is ample evidence of a widespread fibrous mesh slowly strangling the circulation and inexorably binding resilient muscles, tendons, capsules and fasciae down to the unyielding skeleton. Parallel with the decalcification there is a degeneration and absorption of fat and muscle tissue and in the worst cases the final stage presents rather the picture of mummification. *Stage of oedema*
Stage of sclerosis
Stage of atrophy

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Stage of sclerosis
Stage of atrophy

success in the immediate treatment of crush injuries and burns as well as after extensive operative procedures.

Experimental work is in progress for the development of special refrigeration plant so that cold treatment can be applied easily and accurately to these cases.

The value of static muscle contractions cannot be over-emphasized and except when absolute rest is indicated—for example, for an acute infection or a severed tendon—the patient should be taught to contract all his muscle groups at regular intervals; joint movement will be prevented by the dressing. *Static contractions*

(2) Curative

In the early stages of the established swelling the same routine should be observed. The limb should be put at rest in a pressure dressing, with splintage, and elevated. Static exercises should be commenced and fluid drainage encouraged by gentle massage each day. At this stage small anodal galvanic currents of up to 0.5 milliampere, applied twice daily, assist the movement of the fluid.

As the oedema subsides the dressings may be released. More active exercise therapy is then instituted and may be supplemented by massage and wax baths, but adequate periods of rest in splintage must be maintained until pain, discomfort and swelling are absent. If at this stage the circulatory disorder persists, as shown by the cold blue extremity with a poor arterial input, then sympathectomy should be considered. The results are best when this operation is done early and are often disappointing when it is tried in the old-established case. *Physiotherapy*
Sympath-ectomy

All local "irritator" foci should be eliminated and, surgically, this may be combined with the sympathectomy. At all stages competent physiotherapy must accompany the surgical treatment. *Removal of local foci*

[References to other titles are given under Oedema—Traumatic, in the Index Volume. The subject is also dealt with in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 268.]

OESOPHAGUS

By P. R. ALLISON, CH.M., F.R.C.S.
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1. ANATOMY

247.] The oesophagus extends from the cricoid cartilage, at the level of the transverse process of the sixth cervical vertebra, to its entry into the stomach in the abdomen, at the level of the tenth thoracic vertebra. The oesophagus is 23-25 centimetres long and its entrance into the stomach is 38-42 centimetres from the upper alveolar margin. It is described by the anatomists as consisting of a cervical portion, a thoracic portion and an abdominal portion, but for surgical purposes it is enough to divide it into cervical and thoracic segments. Physiologically it is better considered as consisting of *Segments* an upper third and a lower two-thirds. These two segments are differently constructed and have different nerve supplies.

The oesophagus is a muscular organ which is covered by a condensation of fascia known as the fascia propria of the oesophagus. At its lower end this fascia blends with the fascia on the deep aspect of the diaphragm—the phreno-oesophageal ligament. In its upper third the muscle is striated, but in the lower two-thirds this is replaced by non-striated involuntary muscle. It consists of an outer longitudinal layer and inner circular fibres, but careful dissection shows that there are many oblique and spiral muscle fibres as well. At the upper end of the oesophagus the circular muscle fibres are *fascia propria*

continuous with the cricopharyngeal muscle. but the longitudinal muscle splits at the back, curves forwards and is attached to the posterior aspect of the cricoid cartilage.

*Weaknesses
in posterior
wall*

At the junction of the pharynx and oesophagus there are two relatively weak places on the posterior wall, the one between the oblique and transverse fibres of the inferior constrictor of the pharynx and the second between the transverse fibres of the inferior constrictor and the longitudinal fibres of the oesophagus as they curve forwards to the cricoid. At the lower end both longitudinal muscle fibres of the oesophagus are continuous with the longitudinal muscle fibres of the stomach. It seems unlikely that the oesophagus to the pleura or bronchi are

sional muscle fibres of any functional significance.

Lining

*Racemose
mucous
glands*

The oesophagus is lined by stratified squamous epithelium on a fibrous tissue layer and beneath this is the muscularis mucosae which is better developed in the lower part of the organ. The mucous membrane contains numerous racemose mucous glands, but it is not uncommon to find patches of gastric mucous membrane, particularly in the upper third. Some authorities claim that on careful serial section of the oesophagus gastric glands may be found in as many as 55 per cent of those examined. The submucous layer of connective tissue is relatively abundant and loose, so that free movement of the mucous membrane occurs over the muscle. When the organ is contracted the mucous membrane is thrown into longitudinal folds, but when it is distended the mucous membrane becomes quite smooth. During operation on the oesophagus it is very easy to divide the muscle coat and to resect it as a sleeve, leaving the mucous membrane intact. The abundant submucous layer also allows the easy enucleation of simple tumours from the muscle layer without injury to the mucous membrane.

*Easy division
of muscle
coat*

*Nerve
supply*

The upper third of the oesophagus receives its nerve supply from the recurrent laryngeal nerves. The main trunks of the vagus nerves join the oesophagus immediately below the arch of the aorta and form a plexus on its walls. The oesophagus also receives abundant sympathetic nerves from the thoracic sympathetic chain.

(1) Blood supply

The blood supply to the upper part of the oesophagus is mainly from the inferior thyroid arteries, while below there are branches from the aorta itself. In its lower part it also receives arteries from the left gastric and from the inferior phrenic arteries. The anastomoses in the oesophageal wall are adequate to maintain its nutrition in most circumstances. Modern surgery has shown the conception of the oesophagus as relatively avascular to be inaccurate.

Venous drainage

The venous drainage of the oesophagus is into the thyroid veins above, but mainly into the vena azygos system. In the lower part the venous plexus of the oesophagus is partly drained by the vena azygos major and minor and partly by the left gastric vein. In this area, therefore, there is a communication between the portal and systemic venous circulations, and this becomes of great importance when portal hypertension exists.

(2) Lymphatic drainage

The lymphatic plexuses of the oesophagus drain primarily to the lymphatic glands of the posterior mediastinum and above to those at the root of the neck. They communicate freely with the lymphatic glands draining the lungs around the major bronchi and trachea. There is a strong lymphatic flow from the whole of the lower two-thirds of the oesophagus downwards through the hiatus of the diaphragm into the left gastric group of glands, the supra-pancreatic glands and the glands around the coeliac axis. *Course of lymphatic flow*

(3) Cervical oesophagus

The cervical oesophagus lies in front of the spine and anterior spinal muscles from which it is separated by loose connective tissue. In front of it is the trachea and thyroid gland, and between it and the trachea the recurrent laryngeal nerve passes up to the larynx. On each side of it lies the common carotid artery and the internal jugular vein in the carotid sheath.

(4) Thoracic oesophagus

In the superior mediastinum the oesophagus is in relation on each side to the mediastinal pleura from which it is separated on the left by the left subclavian artery, the thoracic duct and the arch of the aorta and on the right by the arch of the vena azygos major.

Below the arch of the aorta the oesophagus is crossed by the right pulmonary artery and the left main bronchus. Below this it lies behind the pericardium, the left auricle and the diaphragm. On its left side is the descending thoracic aorta, but in the lower part of the chest the oesophagus comes forward to the left in front of the aorta and is in direct relation with the mediastinal pleura. On its right side is the right lung and the mediastinal pleura. This part of the oesophagus lies in front of the spine above and the descending aorta below. The right intercostal arteries pass between the oesophagus and the spine.

The lower end of the oesophagus passes through an almost vertical tunnel where it is enveloped by the crura of the diaphragm except for a small triangular area in front which is covered by peritoneum. It passes immediately from its bed in the diaphragm into the stomach and so in the normal position during life there is practically no such thing as an abdominal oesophagus. It would be more accurate to describe this segment as the diaphragmatic oesophagus. This arrangement is confirmed by oesophagoscopic examination when the instrument is found to pass immediately into the stomach after traversing the diaphragmatic pinchcock. *Lower end of oesophagus*
Diaphragmatic oesophagus

2. PHYSIOLOGY

When food enters the oesophagus by the sudden relaxation of the cricopharyngeus, it does so under fairly considerable pressure. The act of swallowing can be compared to the act of coughing inasmuch as an explosive force is produced by the contraction of the cricopharyngeal sphincter at the same time as that of the pharyngeal muscles followed by its sudden relaxation. In patients who have a cervical oesophagostomy it is possible to estimate the force of this mechanism by inserting a vertical glass tube into the oesophagostomy. When the patient swallows fluid he can raise it to a height of $2\frac{1}{2}$ feet in the *Act of swallowing*
Force of swallow

vertical tube before incompetence of the cricopharyngeus occurs with regurgitation into the mouth. If the oesophagus is removed and replaced by an inert rubber tube the force of swallowing alone is enough to make fluids reach the stomach. In normal circumstances, however, the act of swallowing produces a peristaltic wave in the oesophagus which catches up with the bolus usually towards the lower end of the oesophagus and forces it through into the stomach. The continued presence of a bolus in the oesophagus may, however, produce secondary peristaltic waves.

Peristaltic
wave

Mechanism of the gastro-oesophageal junction

The mechanism of the gastro-oesophageal junction has been the subject of much dispute. It has already been stated that the force of swallowing alone would lift a column of fluid to a height of $2\frac{1}{2}$ feet. It is obvious, therefore, that this force alone is enough to propel fluid into the stomach even if the oesophagus is inert and even if the patient should be turned upside down. The peristaltic movement is more important for the solid bolus. There is no physiological or anatomical evidence for a sphincter at the junction of the oesophagus and stomach, and it seems that the circular muscle fibres here must relax during the phase of inhibition of the peristaltic wave. The oblique entrance of the oesophagus into the stomach does form something of a valve mechanism so that when the stomach is distended the cardia tends to collapse. It has been shown that the force necessary to drive fluid from the stomach to the oesophagus is three times that required to drive it from the oesophagus to the stomach even in the excised organs. In life this mechanism is still further augmented by a contraction of the crura of the diaphragm. During inspiration the bolus is held up at the diaphragmatic pinchcock and is allowed to pass through during expiration. If the peristaltic wave in the oesophagus reaches the diaphragm when it is in the phase of inspiration some pressure is developed in the lower end of the oesophagus and dilatation occurs. Radiologists have called this the phrenic ampulla. Although it is difficult to prove, it seems likely that there must be a close co-operation between the diaphragm and the intrinsic mechanism of the oesophagus in the normal state of health, and it may be that interference with this co-operation may result in dysphagia.

No evidence
of sphincter

Contraction of
crura of
diaphragm

Phrenic
ampulla

The importance of the mechanism is not only in allowing food to pass from the oesophagus to the stomach, but also in the prevention of regurgitation of acid contents from the stomach into the oesophagus. Whatever may be the factors in the stomach which prevent the gastric mucous membrane from being digested by its own juices, these factors certainly do not operate in the oesophagus. The frequent presence of acid in this area in cases of persistent vomiting may cause temporary ulceration. When anatomical defects allow the constant bathing of the oesophagus by regurgitated acids from the stomach chronic ulceration is likely to occur.

3. THE SURGICAL APPROACH TO THE OESOPHAGUS

(1) Cervical oesophagus

The cervical oesophagus may be approached from either side of the neck, but if a choice exists it is probably better to use the left side. A collar-shaped incision may be used (Fig. 172) or one along the anterior border of the sternomastoid muscle. The sternomastoid may be retracted outwards with or without

collar-shaped
incision

division of the sternal head. The cervical fascia is divided and the middle thyroid vein divided between ligatures. The omohyoid muscle is retracted upwards or divided and the dissection carried backwards to the spine between the oesophagus and trachea on the inside and the carotid sheath on the outside. Care should be taken not to injure the recurrent laryngeal nerve. The

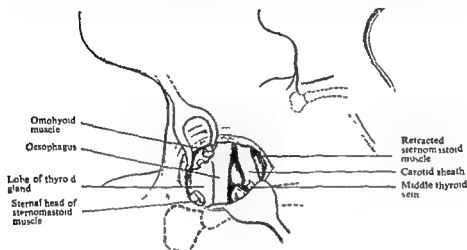


FIG. 172 — Exposure of cervical oesophagus. *Inset shows neck incision*

oesophagus in the neck is very easily separated from the prevertebral muscles but its attachment to the trachea is closer and may in places require sharp dissection. If further exposure of the cervical oesophagus is necessary the inner third of the clavicle should be resected. When the connective tissue layers around the oesophagus have once been entered, it is not difficult to free the oesophagus from a cervical incision as far down as the arch of the aorta.

(2) Thoracic oesophagus

Exploration of any part of the thoracic oesophagus is best effected by a long incision along the ribs, with or without the resection of a whole rib or a posterior segment of one or two ribs. Such a long thoracotomy opening gives the best exposure of any part of the oesophagus with the minimum amount of deformity of the chest wall afterwards.

(a) Approach from right side

The upper part of the thoracic oesophagus is probably best approached from the right side after resection of the fourth or fifth rib beneath the scapula. The oesophagus here lies immediately beneath the mediastinal pleura. If it should be necessary the arch of the vena azygos major may be divided between ligatures. The approach to the lower two-thirds of the oesophagus on the right side is by similar incision after resection of the seventh or eighth rib.

(b) Approach from left side

From the left side the incisions are similar but the approach to the oesophagus in its upper part is not so easy on account of its relation to the left subclavian artery, but this may be displaced forwards and so a fairly satisfactory

Upper part

*Middle third**Lower third**Cardia and
stomach*

FIG. 173.—Skin incision for resection of the eighth or ninth rib and exposure of the lower oesophagus.

the costal margin (Fig. 174). It may be still further improved by splitting the diaphragm back towards the hiatus from the anterior end of the incision



FIG. 174.—Extension of incision across the costal margin for exposure of the lower oesophagus and cardiac end of the stomach. The line of incision of the diaphragm is indicated.

(3) Opening of pleura

For all oesophageal work within the chest it is better to open the pleura widely. There is no contra-indication to extending this into the opposite pleura and the peritoneum, so long as the lungs are kept properly inflated and left fully expanded at the end of the operation so that the physiology of respiration is restored to normal. Timid exposure of the oesophagus by resecting small portions of many ribs and reflecting the pleura without opening it gives poor access and no advantages, and leads to technical difficulties.

4. CONGENITAL ABNORMALITIES

(1) Atresia

The commonest form of atresia of the oesophagus is that variety in which the upper part of the oesophagus ends blindly behind the trachea and the lower end communicates above with the trachea or one of the main bronchi and passes from here down to the stomach. Much more rarely the upper segment of the oesophagus may communicate with the trachea or there may be an opening in the posterior wall of the trachea which passes into both upper and lower segments of the oesophagus. Both segments of the

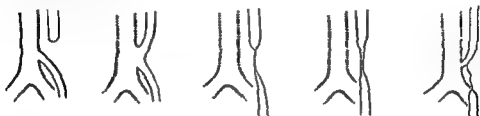


FIG. 175 — Varieties of congenital oesophageal atresia.

oesophagus may be blind and joined to one another by a thin fibrous cord, *Blind segments* or canalization may be complete but inadequate in which case the two segments of the oesophagus may be joined by a fibrous cord in which is a narrow lumen (Fig. 175).

(a) Symptoms

The symptoms of atresia of the oesophagus are quite typical and should be recognized during the first 24 hours of life. As soon as the child takes a feed it goes blue and chokes and the feed is returned. In such circumstances it should not be assumed that the child is merely obstinate, but a well-greased Jacques's rubber catheter should be passed down into the oesophagus and the presence of atresia confirmed. When the diagnosis has once been made a search is necessary for other congenital abnormalities such as imperforate anus, spina bifida and congenital heart disease. Many other congenital associated abnormalities have been described, but these are often not possible to diagnose during life, for example, atresia of the duodenum or bile ducts, absence of ureter, renal abnormalities and genital abnormalities. The presence of many of these may be incompatible with life even if atresia can be relieved. *Use of catheter* *Other congenital abnormalities*

(b) Diagnosis

The diagnosis should be suspected from the history of choking and cyanosis associated with feeding. It is confirmed by the passage of a rubber catheter into the pharynx and oesophagus. In no circumstances should the child be given a barium swallow, for barium entering the lungs produces a broncho-pneumonia and a certainly fatal result. The presence of a tracheo-oesophageal fistula is proved by the distension of the stomach with gas and also the detection of acid in the mouth which has come up from the stomach through the trachea during coughing and choking. *Barium swallow contraindicated* *Tracheo-oesophageal fistula*

Middle third

Lower third

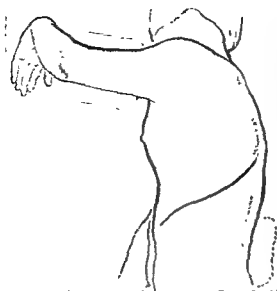
Cardia and
stomach

FIG. 173.—Skin incision for resection of the eighth or ninth rib and exposure of the lower oesophagus.

the costal margin (Fig. 174). It may be still further improved by splitting the diaphragm back towards the hiatus from the anterior end of the incision.



FIG. 174.—Extension of incision across the costal margin for exposure of the lower oesophagus and cardiac end of the stomach. The line of incision of the diaphragm is indicated.

(3) Opening of pleura

For all oesophageal work within the chest it is better to open the pleura widely. There is no contra-indication to extending this into the opposite pleura and the peritoneum, so long as the lungs are kept properly inflated and left fully expanded at the end of the operation so that the physiology of respiration is restored to normal. Timid exposure of the oesophagus by resecting small portions of many ribs and reflecting the pleura without opening it gives poor access and no advantages, and leads to technical difficulties.

4. CONGENITAL ABNORMALITIES

(1) Atresia

The commonest form of atresia of the oesophagus is that variety in which the upper part of the oesophagus ends blindly behind the lower end of the trachea and the lower end communicates above with the trachea or one of the main bronchi and passes from here down to the stomach. Much more rarely the upper segment of the oesophagus may communicate with the trachea or there may be an opening in the posterior wall of the trachea which passes into both upper and lower segments of the oesophagus. Both segments of the



FIG 175 —Varieties of congenital oesophageal atresia.

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(c) Treatment

If the diagnosis can be made in the first 2 or 3 days of life there is some hope for the child's survival with adequate surgical treatment. The factors which militate against success are delay in diagnosis, the presence of other congenital abnormalities, and the presence of barium in the lungs following the energetic, but ill-advised, enthusiasm of the radiologist or physician.

Operation.—Under the very strictest aseptic conditions a fine endotracheal tube should be passed to maintain inflation of the lungs with oxygen during the operation. No anaesthetic agent is administered. The right chest is opened through an incision passing from the spine forwards under the angle of the scapula. The scapula is elevated and the posterior end of the third, fourth, fifth and sixth ribs resected. The parietal pleura is carefully stripped without damaging it until the posterior mediastinum is exposed. If an extrapleural approach is successful the chances of survival are increased. The anaesthetist should pass a fine rubber catheter through the mouth into the pharynx, and this helps to locate the upper blind end of the oesophagus which is carefully freed from its surroundings. The arch of the vena azygos is divided between ligatures and the lower segment of the oesophagus isolated where it passes into the trachea. A ligature of fine silk should be passed round this and tied just at the point of entrance into the trachea or main bronchus. This ligature should be left long and used for traction. This is pulled firmly upwards as the upper segment of the oesophagus is united to the lower segment before the lumen of either has been opened; 2 or 3 stitches of interrupted catgut are sufficient for this layer. A small incision is then made in each segment of the oesophagus and 2 or 3 fine interrupted catgut sutures may then be passed down into the lower segment and the anastomosis completed by interrupted sutures over this tube. Only when this suture has been completed should the oesophagus be divided from its attachment to the trachea. During closure of the chest slight positive pressure is applied by the anaesthetist to ensure full distension of the lungs. The catheter in the pharynx may be left in place in order to re-establish fluid balance for 24 hours, or it may be removed and a gastrostomy performed. By this time the anastomosis is probably surrounded by mediastinal tissues and pleura and it is safe to remove the catheter. The operation should be assisted by a small blood transfusion of 100–250 cubic centimetres of carefully matched blood. The child should be returned to an oxygen tent and aspiration of fluid from the right chest may be necessary during the post-operative phase. Although this is a very formidable operation associated with a high mortality it is the only possible line of attack, for any less severe intervention such as gastrostomy is only followed by the aspiration of feeds into the lung and inevitable death.

(2) Compression of the oesophagus by abnormal arteries

The arch of the aorta may sometimes pass between the oesophagus and the spine, compressing the oesophagus against the trachea. Sometimes an abnormal subclavian artery may complete a ring between which the oesophagus and the trachea are compressed (Fig. 176). With a right-sided aortic arch the left subclavian artery may pass between the spine and the oesophagus and compress it, or occasionally the aortic arch may be double and form a

Adverse factors

Endotracheal intubation

Incision

Rib resection

Freeing of upper blind end

Incision of each segment

Full distension of lungs

Blood transfusion

High mortality

circle round the oesophagus and trachea (Fig. 177). If the arch of the aorta passes behind the oesophagus a vascular ring may be completed by a large patent ductus arteriosus. With a normally placed aortic arch it sometimes happens that the right subclavian artery passes from left to right between the

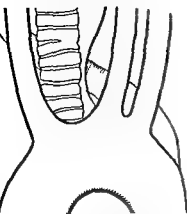


FIG. 176.—Abnormal right subclavian artery causing dysphagia lusoria

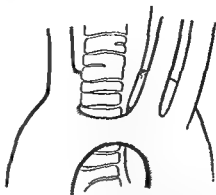


FIG. 177.—Double aorta causing compression of trachea and oesophagus.

trachea and the oesophagus. Any of these abnormalities may lead to dysphagia, and the term dysphagia lusoria is now used to include them all. Although the abnormality is a congenital one it is rare for symptoms to occur before the age of 50 years after which arteriosclerotic changes have taken place. Examples of obstruction in early life, however, have occurred and it is possible, if a diagnosis can be made, to relieve some of these by surgical division of the constricting vessel.

3) Congenitally short oesophagus

A congenital failure of longitudinal development of the oesophagus is described. When this occurs a variable proportion of the stomach is found in the posterior mediastinum. This, in itself, might not give rise to symptoms if it were not for the breakdown of the mechanism of the cardia with peptic ulceration of the lower end of the oesophagus. Many of the cases described as congenitally short oesophagus do not come under this heading, but are examples of congenital maldevelopment of the diaphragmatic hiatus with a sliding hernia of the fundus of the stomach. In these circumstances peptic ulceration of the oesophagus occurs and in a new-born child tends to produce a rapidly ascending fibrosis of the oesophagus with stricture formation.

The fibrosis of the oesophagus leads to lack of elasticity and fixation of the stomach in the posterior mediastinum. Vomiting or regurgitation may be noticed within the first week of life and has to be diagnosed from congenital oesophageal stenosis and pyloric stenosis. A skiagram after swallowing a little Lipiodol will clarify the doubt. Haematemesis from peptic ulceration has been noted in the first few days of life.

Treatment

There is no satisfactory immediate treatment for congenitally short oesophagus, but some relief of symptoms may occur if the child is nursed propped

up after its meals so that regurgitation into the lower end of the oesophagus is not so free.

Operation.—In children with congenital hernia (Fig. 178), if the diagnosis can be made before permanent fibrosis and shortening of the oesophagus occur, operation is urgently called for. Under endotracheal anaesthesia the left pleura should be widely opened through an incision in the sixth interspace with or without excision of the posterior end of the rib.

The lung is retracted forwards and, after a little local anaesthetic is injected into the vagal plexus, the posterior mediastinum is opened and a thread passed round the lower end of the oesophagus where it joins the stomach. A small hole may then be made in the left dome of the diaphragm, the tape or silk round the cardia passed

Incision



(a)

FIG 178—Skilagrams taken (a) before and (b) after reduction of a sliding hernia with a para-oesophageal pouch in a child aged 10 months.



(b)

through the hiatus of the diaphragm and back into the chest through the hole in the diaphragm. Traction on this will then reduce the hernia into the abdomen when the diaphragmatic crura can be lightly stitched. The stitches in the diaphragm should be inserted behind the oesophagus. The wall of the oesophagus may be lightly sutured to the diaphragmatic crura at the point where it passes through the hiatus. When fibrosis and permanent shortening of the oesophagus have occurred the child should be treated by oesophagoscopy dilatation of the stricture until it is old enough to withstand the formidable operation of excision of the lower end of the oesophagus and anastomosis to a Roux loop of intestine with exclusion of the stomach.

(4) Oesophageal web

When this rare abnormality occurs it is found at the level of the bifurcation of the trachea. If the web is complete enough to cause symptoms it is associated with intermittent attacks of dysphagia. These attacks are caused by the lodging of a solid bolus at the level of the web blocking up the lumen. The child may be described as having a small swallow and may reach adult life without undue attention being paid to the trouble. The patient may appear for treatment because of complete obstruction and only when a foreign body is removed through the oesophagoscope may the web be found. The only example of this abnormality occurring in the author's experience was in a young student in whom a bolus of food became impacted at the web. After the bolus was removed the oesophagoscope was passed through the web and it was ruptured. This occurred with only a very slight oozing of blood and was followed by complete relief of symptoms.

5. DIVERTICULA

(1) Congenital

Most diverticula which occur in the oesophagus are acquired, but a congenital diverticulum is found occasionally and results from the spreading out of entodermal cells from the primitive alimentary canal. These cells may become completely separated from the oesophagus and form a mediastinal cyst or they may retain their communication with the oesophageal lumen and form a diverticulum (Fig. 179). Such a pouch is often lined not only by squamous epithelium of the oesophagus, but also contains gastric heterotopia, and it has occasionally been the seat of peptic ulceration. These diverticula are often



FIG. 179.—Congenital diverticulum of the oesophagus infected and filled with pus

These diverticula are often

occur in the posterior mediastinum, more often in the lower half than the upper half. The patient may complain of regurgitation of oesophageal contents, slight dysphagia and a feeling of fullness in the chest after meals.

If the pouch is giving rise to symptoms which are severe enough it should be removed. Although the pouch may project into the right pleural cavity, it is probably wise to explore the oesophagus from the left side in most cases. The cyst can be dislocated into the left pleural cavity, excised and the hole in the oesophagus sutured.

(2) Acquired

A diverticulum may occur on the anterior aspect of the oesophagus at the level of the bifurcation of the trachea or just below. In the first instance this is believed to be a traction diverticulum and to be caused by fibrosis of lymphatic glands. It is usually an incidental radiographic finding (Fig. 180), and does not cause symptoms, but occasionally it may enlarge by pulsion and become dependent, when it is possible that food retention and infection may occur. Other diverticula are probably of the pulsion variety and are associated with muscular weakness of the oesophageal wall. It is possible that these are a predisposing cause of spontaneous rupture of the oesophagus. They may be associated with dyspepsia, epigastric pain and vomiting, but little is known about their importance in the production of



FIG. 180.—Multiple acquired oesophageal diverticula in a patient who complained of slight dysphagia, epigastric pain and vomiting.

symptoms. They may be multiple, and if large and dependent and apparently causing symptoms they may be excised and the muscular wall of the oesophagus sutured.

6. RUPTURE

(1) Spontaneous

Spontaneous rupture of the oesophagus, drinking and vomiting, is more common in men than in women. The rupture is usually gradual and has been common since the introduction of over-drying

THE OESOPHAGUS

the oesophagus is as very rare accidentally as rupture in the oesophagus, in the oesophagus, although the rupture is as

the excessive rupture is more common at the lower part of the oesophagus, rather than at the upper part.

Fibrosis of lymphatic glands

Spontaneous rupture

Excision

Onset of symptoms

also been reported as a result of seasickness, of lifting heavy weights or of crushing injuries involving the chest. When the oesophagus ruptures a longitudinal tear is usually produced and this may extend into the mediastinum or into one or both pleural cavities. Occasionally transverse tears have occurred and the ends of the severed oesophagus may be separated in the mediastinum.

(a) Signs and symptoms

Sudden, severe pain in the chest and a sensation of pressure on the heart and lungs have usually occurred. The pain may, however, be felt mainly in the *Abdominal* abdomen, and is usually described as excruciating. The patient is restless, *pain* anxious, pale and slightly cyanosed. The pulse is usually rapid and faint. The most striking physical sign is uniform board-like rigidity of the upper abdomen, and this may extend over the whole of the abdomen and be very suggestive of a perforated gastric ulcer. Emphysema into the mediastinum may occur and shows at the root of the neck. The early onset of shock is helpful in distinguishing rupture of the oesophagus from rupture of an ulcer in the abdomen. Examination of the chest may reveal a flat note with absent air entry on one or both sides, and when any doubt exists an x-ray examination of the *Radiography* chest and the abdomen is imperative. The diagnosis is confirmed by aspiration of the chest when air and gastric contents may be obtained.

(b) Differential diagnosis

The clinical picture must be distinguished from perforated gastric or duodenal ulcer, perforated peptic ulcer of the oesophagus or perforation of an incarcerated diaphragmatic hernia. The most important thing is to decide whether the lesion is above or below the diaphragm, for on this depends whether a laparotomy or a thoracotomy is to be performed. If it is not possible to be certain about this, the safest plan is to make an abdomino-thoracic incision under positive pressure anaesthesia and explore both the upper abdomen and posterior mediastinum. If rupture of the oesophagus is diagnosed an immediate thoracotomy should be performed. This should obviously *Thoracotomy* be done through the pleural cavity which is infected. The longitudinal tear in the oesophagus should be closed by a double layer of fine sutures, the pleura should be sucked dry and the chest closed with an under-water drain to ensure complete expansion of the lungs. It is not necessary to perform a gastrostomy, but it is wise to see that the stomach is completely emptied.

(2) Traumatic

The commonest cause of injury of the oesophagus leading to perforation is the injudicious use of the oesophagoscope. During this operation the instrument may be passed through the weak posterior wall of the pharynx immediately above the crico-pharyngeal sphincter and this is the accident most likely to occur. When the instrument has once passed into the oesophagus perforation of the wall may occur during the manipulation of an impacted foreign body, or the oesophagoscope itself may be passed through a weak part of the wall above an inflammatory or malignant stricture *Causes of rupture*

(a) Signs and symptoms

The patient complains of severe pain in the chest and back made worse by deep breathing or coughing. He may be very dyspnoeic if a pneumothorax has occurred, and surgical emphysema develops as soon as he starts to cough. If

the patient has been under a general anaesthetic and coughing has not occurred the pulse may become rapid and thin, the skin cold and clammy with sweat and the abdomen rigid before any emphysema develops. The diagnosis must be made and treatment started even in the absence of surgical emphysema or pneumothorax. If doubt exists the patient should be given a small quantity of Lipiodol to swallow; a skiagram will then confirm the presence of an oesophageal tear and is useful in deciding the size of the lesion.

Radiography

(b) Treatment

Immediate repair of the perforation should be undertaken. When the cervical oesophagus is involved an incision through the deep fascia along the anterior border of the sternomastoid with the division of the sternal head of that muscle and retraction of the carotid sheath outwards gives an excellent exposure. In the acute case the rent may be sutured with double rows of catgut, penicillin applied and the wound closed with a small rubber drain. The patient is allowed fluids from the beginning and the wound usually heals by first intention. If operation has been delayed and an abscess has formed it should be incised and drained, and all sloughs should be removed. Systemic penicillin and parenteral fluids should be administered for 3 or 4 days before gastrostomy is considered because it is often not necessary to do this and the oesophageal fistula closes quite quickly. If it persists, however, and not enough food can be taken by mouth, it may be necessary to do a gastrostomy some days after the abscess has been drained.

Closure

Gastrostomy to be delayed

(i) *Thoracotomy.*—If the injury is in the thoracic part of the oesophagus an immediate thoracotomy on the side of the opening should be done. The side may be indicated either by the presence of a pneumothorax or by a skiagram after the swallowing of Lipiodol. A long incision beneath the angle of the scapula with resection of the whole of the sixth or seventh rib will give a good exposure of almost any part of the oesophagus. The lung should be retracted forwards and the posterior mediastinal pleura widely opened along its whole length. If operation is performed within a few hours of the accident, the oesophagus may be repaired by a double row of interrupted catgut stitches so that the resulting suture-line is transverse. In one patient in whom perforation had occurred above a fibrous stricture, the tear was enlarged longitudinally to include the stricture so that when it was repaired transversely the tear and the stricture were cured at the same time. Penicillin should be applied locally, the mediastinal pleura left widely open and the chest closed with an underwater drain to ensure complete expansion of the lung. After 24 hours the drain may be removed, and if further pleural fluid accumulates it may be aspirated. There is no need to perform gastrostomy and the patient may be

Indications

Operative procedure

Aspiration of fluid

but care should be taken to remove all sloughs. Penicillin-sulphonamide powder should be applied liberally and a longer drainage tube should be used to pass through the pleura and up along the posterior mediastinum.

(ii) *Prognosis.*—When early operation is effectively carried out and in the absence of underlying malignant disease of the oesophagus the prognosis is very good.

7. ACUTE OESOPHAGITIS

Acute oesophagitis may arise from a pharyngeal infection spreading down the gullet. It causes soreness behind the sternum and an awareness of food passing down. It may also arise from long-continued vomiting or from acid digestion in long and debilitating illnesses in which acid regurgitates easily from the stomach. These conditions are of more medical than surgical importance. Radiotherapy for bronchial or oesophageal carcinoma may also be a cause. *Causative factors*

The swallowing of hot or corrosive fluids is one of the most serious causes of acute oesophagitis. The lesion may vary from a superficial lesion to deep destruction as in burns of the skin. If not fatal in the early stages, the formation of a long, complicated and dense fibrous stricture is next to be feared. When possible this should be prevented rather than left to develop and then treated.

Treatment

An immediate gastrostomy should be performed in order to maintain the patient's fluid balance and nutrition. If the general condition is very bad it may be necessary to delay gastrostomy for a few days during which intravenous fluids are given. After a week or 10 days, when the acute reaction has begun to settle, oesophagoscopy should be performed under intravenous anaesthesia in order to determine the extent of the damage. The greatest care must be used to prevent further trauma to the already friable oesophagus. After this, well-greased, soft rubber catheters may be passed each day to maintain a good lumen. If reaction in the oesophageal wall obstructs the catheter at any time force must not be used but an oesophagoscopy performed and the tube passed under direct vision after careful dilatation. After this, blind intubation may be repeated.

As an alternative to the intermittent dilatation by catheterization, it has been found useful to open the oesophagus at the root of the neck on the left side and to pass a medium-sized stomach tube through the opening into the stomach and leave this in position. If this is done the patient may be fed through the stomach tube and the gastrostomy allowed to close. One tube then fulfils the double function of carrying food and maintaining continuous dilatation of the oesophagus. The tube should be removed each day and immediately replaced by a similar sterilized one. After a few weeks the tube may be removed and replaced by a pad during meal time, so that the patient may have the satisfaction of swallowing a fluid or very soft diet by mouth. If the tube is replaced between meals the oesophagus is never given the chance of forming a tight stricture. Only experience can decide when to abandon the cervical oesophagostomy and rely on the occasional blind passage of bougies. In the first few weeks after recourse to blind bougination this may have to be done every 3 or 4 days, but the time interval can be extended to weekly, then monthly, treatments until the oesophagus is completely epithelialized and treatment no longer necessary. *Cervical oesophagostomy*
Removal of stomach tube
Blind bougination

8. OESOPHAGEAL VARICES

The veins of the oesophagus anastomose with other mediastinal veins and drain into the vena azygos major and the superior and inferior vena azygos

minor. At the lower end of the oesophagus, however, some of the veins drain into the left gastric vein and so into the portal circulation. At the cardia, therefore, there is a free communication between the portal and systemic circulations by way of the oesophageal veins.

*Portal
obstruction*

Obstruction to the portal circulation may occur within the liver, as for example in cirrhosis of the liver, or outside the liver as in portal or splenic

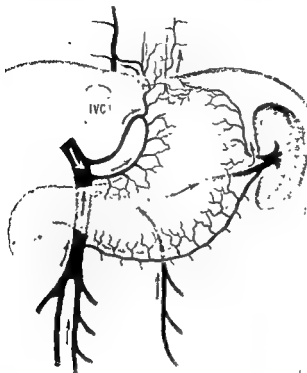


FIG. 181—Diagram to show the anastomotic channels in portal obstruction

vein thrombosis. Such obstruction leads to increased pressure in the portal system and to dilatation of such collateral channels as will assist the blood back to the heart. The flow of venous blood in the stomach and cardia and from the spleen is normally towards the liver, but in portal obstruction the current is reversed and blood coming into the portal vein from the intestines is shunted along the left gastric and splenic veins towards the cardia (Fig. 181); it is carried to the heart from the oesophageal veins through the azygos veins. These channels become very greatly dilated and as the pressure in them is increased they

are liable to rupture. Those beneath the mucous membrane of the stomach and oesophagus have the least support and are most liable to trauma and peptic ulceration. Such accidents lead to very severe and often fatal haematemesis.

Treatment

Various forms of treatment have been devised from time to time to diminish the dangers of bleeding in these patients, but it cannot be said that the problem has been solved. Splenectomy may be curative only if the obstruction is in the splenic vein itself. Splenectomy with ligation of the left gastric vein is an incomplete operation, but it may reduce the venous pressure temporarily in a patient who is desperately ill with recurrent haematemesis. Injection of the oesophageal veins through the oesophagoscope does not cause thrombosis and, if it did, it seems likely that it would increase still further the obstruction to the return of blood to the heart. A logical approach is to relieve the portal tension by anastomosing the splenic to the renal vein or the portal vein to the inferior vena cava, but this must be discussed in detail elsewhere.

Splenectomy

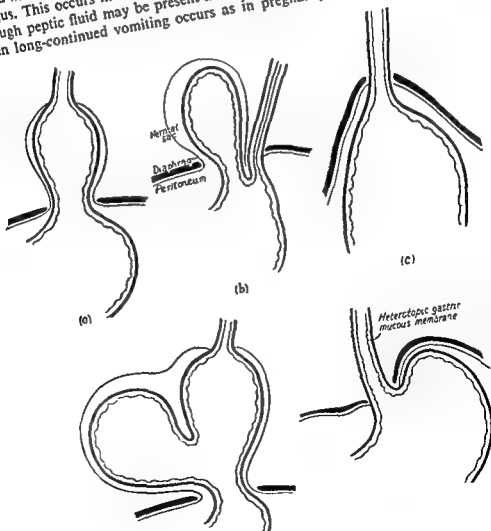
PEPTIC ULCERATION

9. PEPTIC ULCERATION OF THE OESOPHAGUS

With the exception of malignant disease, peptic ulceration of the oesophagus is the commonest cause of dysphagia in patients over the age of 50 years. It is also an important cause of feeding difficulties in infants and young children, but is less frequently seen in the intermediate age-groups.

(1) Aetiology

Peptic ulceration of the oesophagus occurs from an abnormally acting cardia which allows easy regurgitation of the stomach contents into the oesophagus. This occurs most often in sliding diaphragmatic hernia (Fig. 182). Enough peptic fluid may be present in the oesophagus to cause ulceration when long-continued vomiting occurs as in pregnancy, when part of the



Predisposing conditions

oesophagus is lined by heterotopic gastric mucous membrane, when excessive fat and general lack of muscle tone cause a bulging deformity at the diaphragm.

the oesophagus. Ulceration is not often found in the common variety of para-oesophageal diaphragmatic hernia, for in this the cardia usually remains below the hiatus and the oblique entry of the oesophagus into the stomach is maintained. When a sliding hernia and a para-oesophageal hernia exist together, however, ulceration of the oesophagus may occur. The condition is only slightly more common in men than in women.

(2) Pathology

Superficial oesophagitis

The presence of peptic fluid in the oesophagus, especially for many hours at a time as during sleep, leads to a well-defined series of changes. Chronic superficial oesophagitis is first seen and is most marked at the lower end of the oesophagus. The mucosa becomes thickened, pale and oedematous and on microscopic section shows the changes of leucoplakia. Small acute erosions then appear; these come and go and may be multiple, varying in diameter from one or two millimetres to one centimetre; they appear as bright-red patches sometimes partly covered by a thin purulent membrane. When chronic ulceration occurs a crater may be formed, but more often there is a dense fibrous reaction in the submucous layer which causes thickening of the wall and narrowing of the lumen. The muscular coat may show some fibrosis and collection of lymphoid and plasma cells, but it is not usually involved in the ulcer. A sliding hernia of the stomach allows the elastic oesophagus to shorten, and when fibrosis has occurred this shortening becomes permanent so that the stomach can no longer be replaced in the abdomen. This picture has led to the conclusion that peptic ulcer of the oesophagus was caused by a congenitally short oesophagus. The opinion now held is that the shortening is usually acquired and that its permanence is the result of ulceration and fibrosis (Fig. 183).

Appearance of erosions

Chronic ulceration

Shortening of oesophagus

Fibrosis and narrowing do not always occur, however, for in some patients the reaction may remain superficial and be associated with weakening of the muscular and elastic layers of the oesophagus. This leads to an abnormally patulous cardia with equally easy passage of food downwards or upwards (Fig. 184).

(3) Symptoms

Dyspepsia

The commonest symptoms are dyspepsia and dysphagia, the former occurring in 74 per cent of patients and the latter in 92 per cent. Dyspepsia may be variously described as heartburn relieved by alkalis and bland foods, wind, eructations or epigastric pain. The symptoms are easily confused with those of cholecystitis and diagnosis is further confounded by the frequent association of peptic ulcer of the oesophagus with ulcers in the stomach or duodenum and with gall-bladder disease. Haematemesis and melaena may occur; hiccup is a rare complaint. Patients may say they vomit, but most often this is regurgitation of oesophageal contents when stenosis is present. When the cardia is patulous they may regurgitate from the stomach without actually vomiting. Loss of weight is in proportion to starvation which in its turn depends on the amount of oesophageal stenosis.

Regurgitation

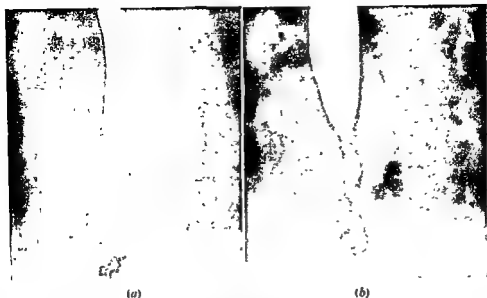


FIG. 183 —Radiographic appearances of peptic ulcer of the oesophagus with sliding hernia: (a) smooth stenosis; (b) irregular stenosis

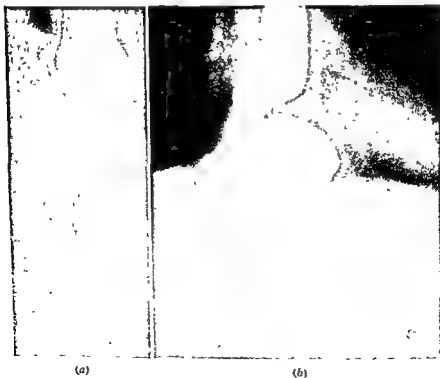


FIG. 184 —Radiographic appearances of peptic ulcer of the oesophagus with sliding hernia: (a) crater; (b) patulous cardia.

(4) Treatment

Palliative measures

Of the adult patients with peptic ulcer of the oesophagus, 90 per cent are over the age of 50 years and the treatment is necessarily influenced by this. It is often possible only to give palliative treatment for, so long as the anatomical abnormality persists, ulceration is liable to be chronic or recurrent. The most important palliative measure is to teach the patient to sleep sitting upright to diminish the regurgitation of acid into the oesophagus during the night. A glass of milk should be given before retiring and a second one kept by the bedside in case he should wake. Meals should be regular but milk may be taken between meals. Strict dieting is of no great value but food should be mechanically and chemically bland and should be masticated thoroughly; for this purpose the teeth should be efficient. It may be necessary to combine the above treatment with occasional dilatation of the oesophageal stricture but if this is done it should be under direct vision through the oesophagoscope and executed with great gentleness. Blind or forcible dilatation is to be condemned.

Dilatation of stricture

Surgical reduction

In the absence of fibrous scarring of the oesophagus at the site of ulceration the ideal treatment is to reduce the hernia surgically. It is not enough to keep the stomach below the diaphragm; the normal physiology of the hiatus must be restored if ulceration is to be cured.

(a) Reduction of sliding diaphragmatic hernia

Rib resection

The stomach and oesophagus should be aspirated clean before operation. A left-sided spinal anaesthetic with or without general narcosis is suitable in most instances. The patient is placed on the right side and the left pleural cavity widely opened after resection of the whole of the eighth rib. It is not necessary to cut across the costal margin. Local anaesthetic may be injected into the mediastinum and into the left phrenic nerve but the latter should not be crushed. The lung is retracted forwards and upwards and the mediastinal pleura is incised behind the lateral pulmonary ligament. The lower end of the oesophagus is exposed and tape passed round it. It may be mobilized by division of the vessels entering and leaving the lower four inches and it may also be helpful to divide the vagus nerves. A radial incision may then be made in the left dome of the diaphragm passing backwards and inwards from the anterior end of the chest incision. Bleeding vessels are stitched and one stitch on each side may be left long for traction. The fingers may then be inserted

Mobilization of the oesophagus

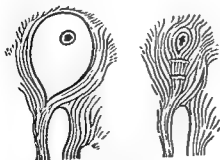


FIG. 185.—Diagrams to show the arrangement of muscle fibres round the hiatus and the suture of these behind the oesophagus in the repair of hiatal hernia.

into the abdomen and passed up into the hiatus to determine the exact anatomy. As the fundus of the stomach is retracted by the tape round the oesophagus the muscular fibres of the hiatus are carefully and thoroughly cleaned. This should be carried well down posteriorly where the fibres from the right crus sweep round to the left to form the posterior boundary of the hiatus. These fibres are usually displaced by the hernia and it is essential that they should be incorporated in the repair if a functional hiatus is to be made. The

peritoneum should then be incised round the margin of the hiatus and the stomach drawn down into the abdomen. The tape round the oesophagus is passed through the hiatus into the abdomen and then back again into the chest through the incision in the diaphragm. Traction on the tape will then maintain the cardia in the abdomen. The two pillars of the right crus are then drawn together behind the oesophagus by two silk stitches (Fig. 185). These should appose the muscle fibres without being tied tightly enough to cut or strangulate them. The fascia propria of the oesophagus is stitched to the fascia of the diaphragm with a few interrupted catgut sutures and the peritoneal cuff on the stomach which formed the hernial sac may be similarly stitched to the under-surface of the diaphragm. The tape is withdrawn from the cardia and the incision in the diaphragm closed. The lungs may then be inflated, any blood aspirated from the pleura, and the chest closed. It is useful to close the chest with an under-water drain in position, and to remove this as soon as a skiagram shows that all air has been expelled from the

Incision of peritoneum



(a)

FIG. 186—Sliding hernia of the stomach with peptic ulcer of oesophagus but no fibrosis* (a) before and (b) after surgical treatment by reduction and radical cure.



(b)

OESOPHAGUS

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pleura. The ideal is to have a skiagram taken on the table and to remove the tube before the patient leaves the theatre.

A successful result in the absence of stenosis is illustrated in Fig. 186. When stenosis of the oesophagus has occurred the fibrosis of the wall makes it unlikely that the hernia can be reduced into the abdomen and left there with a functional cardia. Attempts at extensive mobilization of the oesophagus and tight suturing of the diaphragm only lead to a more severe degree of stenosis, the cutting through of stitches and persistent ulceration. When operation is indicated in the presence of fibrosis, excision of the affected area of the oesophagus is the only effective measure. Continuity should be restored by oesophago-jejunostomy, for a mediastinal anastomosis to the stomach reproduces the deformity which caused the ulceration and is likely to lead to recurrence of symptoms.

(b) *Oesophago-jejunostomy with resection of peptic ulcer of oesophagus*

Pre-operative
preparation

The pre-operative already described for oesophagitis and approach are the same as for the operation already described for oesophagitis and sliding hernia without stenosis except that the incision should extend across the costal margin to the outer border of the rectus abdominis (Fig. 187) At the anterior end of the

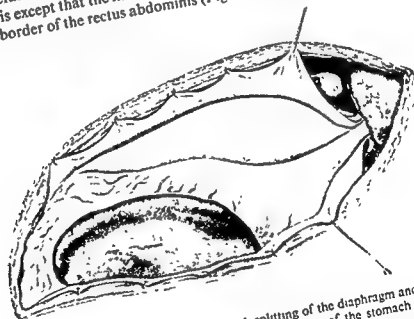


FIG. 187.—Thoraco-abdominal incision with splitting of the diaphragm and suture of this to the edge of the wound showing wide exposure of the stomach and lower oesophagus.

wound an incision is made in the diaphragm which is carried backwards into the oesophageal hiatus. Bleeding vessels in the diaphragm are stitched. One or two of the sutures through the cut edge of the diaphragm are stitched to the edge of the wound and some posterior ones are left long and clips applied to these act as retractors. When this incision is completed there is no need for retractors as the wound opens up like the leaves of a book. The transverse colon and great omentum are drawn up and the first loop of jejunum found. Inspection of the mesentery and its vessels determines the point of section of the intestine which is usually between 5 and 10 inches from the duodeno-jejunal flexure. An incision is made in the mesentery from the intestine at the point selected, and the

Site of
incision

The incision is then continued along the length of the jejunum to the arch, and the division of the mesentery continued until a length of intestine is obtained which would pass easily up to the oesophagus above the site of the lesion (Fig. 188)

FIG. 188.—The making of a Roux loop of jejunum for anastomosis to the oesophagus

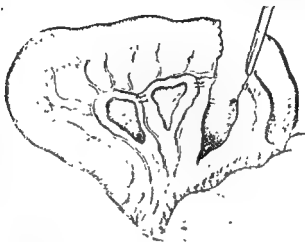


FIG. 189.—Forward retraction of the spleen, stomach and pancreas to make a bed for the intestinal loop. Note the hole in the transverse mesocolon through which the loop will be drawn.

The free end of this loop of intestine is stitched and infolded into itself so that a blind end is formed. A hole is made in the transverse mesocolon and the loop of intestine threaded through this (Fig. 189). The free end of jejunum left in continuity with the duodenum is implanted into the side of the isolated loop

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below the transverse mesocolon (Fig. 190) at a point chosen to avoid tension or kinking. The mesenteries are sutured to prevent internal herniation. The oesophagus is then isolated above the ulcer after division of the mediastinal

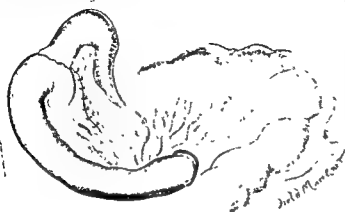


FIG. 190.—T-anastomosis below mesocolon.

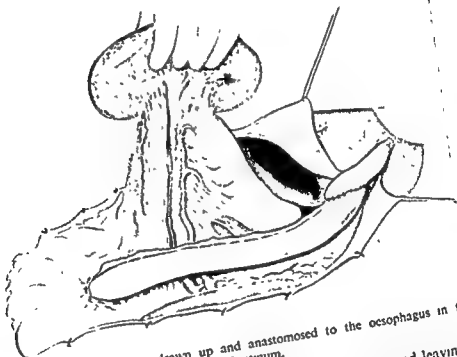


FIG. 191.—Jejunal loop drawn up and anastomosed to the oesophagus in the mediastinum.

Division of
vessels

Removal of
ulcerated
area

pleura. Both vagus nerves are divided; the vessels entering and leaving the oesophagus at this level are divided between ligatures. A light clamp is applied to the oesophagus above the point selected for anastomosis and a crushing clamp just above the ulcer. The ulcerated part of the oesophagus is freed down to and including the cardia itself. Two crushing clamps are applied to the cardia and the viscus is divided between them. The oesophagus is divided between the anastomosis clamp and the crushing clamp above, and the ulcerated area with a portion of the cardia attached is removed. The stomach is sutured and infolded so that it is completely excluded. The lieno-renal ligament is divided and the spleen and pancreas are drawn forwards to expose the

perirenal fat, the adrenal gland and the cupola of the diaphragm. The isolated loop of intestine is then drawn up on this bed behind the pancreas, splenic vessels and fundus of the stomach, to pass through the incision in the diaphragm into the mediastinum, care being taken that twisting of the mesentery of the loop of intestine does not occur. The blind end of the intestine is tucked into the mediastinum behind the oesophagus and the two are united, the end of the oesophagus being implanted into the anterior aspect of the loop of intestine (Fig. 191). The posterior muscle layer of the oesophagus is sutured to the anterior aspect of the intestine by interrupted catgut sutures. An incision is made in the intestine to correspond with the diameter of the oesophagus and a continuous catgut stitch is inserted through the full thickness of both. This is carried right round the circumference. The anterior walls are then reinforced by interrupted catgut stitches so that the mucosal junction is sunk in an "ink-well" of intestine. Penicillin powder is applied and the intestine may be fixed to the cut edge of the mediastinal pleura and the lateral pulmonary ligament. The diaphragm is closed around the intestinal loop and the peritoneum of the intestine is sutured to

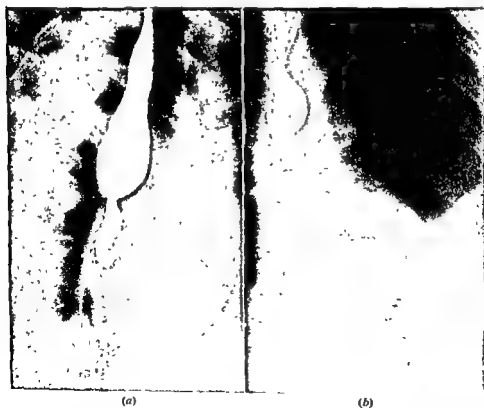


FIG 192.—Skiagrams of peptic ulcer of oesophagus (a) and the anastomosis after excision of the ulcer and exclusion of the stomach (b).

the diaphragmatic pleura. The abdomen and chest are closed in layers, an under-water drain being left in the pleura while the lungs are inflated and closure of the wound completed; the drain may then be removed.

Post-operative management.—The patient should be nursed lying flat for 24 hours and may be given clear drinks as required in small quantities. After 48 hours if there is no abdominal distension and if intestinal movements can be heard, the patient may be given a high-calorie fluid diet and within 5 days should be taking light solids (Fig. 192).

10. SIMPLE TUMOURS OF THE OESOPHAGUS

Simple retention cysts, papilloma, lipoma, fibroma and leiomyoma have all been described as occurring in the oesophagus. They may grow into the lumen and become pedunculated and may prolapse into the stomach. If the pedicle becomes very long the tumour may even be extruded from the mouth by vomiting. All the simple tumours are rare but perhaps the commonest of them to be of clinical importance is the leiomyoma. This tumour occurs during middle age and is most often found in the middle third of the oesophagus or at the lower end. It remains simple and encapsulated but the muscle fibres of the oesophagus over it are prone to become tendinous and to blend with the tumour at what is presumably the site of origin. Typically it forms an ovoid potato-like tumour but it may occasionally encircle the oesophagus as a fibro-leiomatous mass, and this seems to be recorded most often at the cardia when the clinical and indeed the radiological appearances may be mistaken for cardiospasm. In neither variety is there any breach of the mucous membrane.

Leiomyoma

Dyspepsia

Dysphagia



FIG. 193.—Leiomyoma of oesophagus.

(1) Symptoms

The ovoid tumour causes a sense of fullness behind the sternum during swallowing. Poorly masticated solid food may be held up a little but there is usually no notable obstruction. There may be dyspeptic symptoms referred to the epigastrium as with almost any mediastinal tumour. When the whole circumference of the lower end of the oesophagus is occupied by the more extensive type of tumour then dysphagia and the accumulation of frothy mucus in the gullet may be noted.

(2) Diagnosis

Clinical diagnosis is not usually possible for the

history may be indeterminate and physical signs absent. The radiological appearances are of two varieties depending on the morbid anatomy of the tumour. The ovoid submucous tumour causes a smooth permanent indentation of the barium stream, and the length of this corresponds to the length of the soft tissue shadow seen in the mediastinum on the oblique radiograph (Fig. 193). At the lower end of the tumour the barium often forms a crescent in the antero-posterior view, due to its lodging in the sulcus between the tumour and the mucous membrane.

When the tumour encircles the cardia a smooth stenosis is produced in which there are no oesophageal movements. The length of the stricture corresponds to the length of the tumour and there may be gross dilatation of the oesophagus (Fig. 194) above with a sigmoid conformation very like that seen in advanced cardiospasm.

The diagnosis may be supported by oesophagoscopy when a bulging tumour is seen with the mucous membrane stretched over it but not ulcerated or fixed. Oesophagoscopic punch biopsy should not be performed as this may predispose to tearing of the mucosa during operative removal of the tumour.

(3) Treatment

When symptoms justify surgery, and when the general health of the patient permits, the tumour should be removed. For tumours in the upper third the approach should be through the right pleural cavity after resection of the

fifth rib. Any pleural adhesions are divided and the lung is retracted forwards to expose the posterior mediastinum. The tumour can then be palpated. The mediastinal pleura is incised, and if the vena azygos is in the way it may be divided between ligatures. The oesophagus is then isolated above and below the tumour by reflection of the cellular tissues and tapes are passed round for gentle traction. The muscle wall of the oesophagus is then incised over the mass in the long axis of the oesophagus and the tumour is carefully enucleated without injury to the mucosa. If the latter is inadvertently torn it must be sutured from above downwards by a fine catgut stitch. The muscle wall of the oesophagus is then repaired by fine interrupted catgut or silk stitches which should at the same time stop all bleeding. The mediastinal pleura may be



FIG. 194 — Huge leiomyoma of oesophagus encircling the lower end, causing stenosis and great dilatation of the oesophagus.

*Oesophago-
scopy*

*Enucleation
of tumour*

sutured or not at will, but there is no harm in leaving it open and this does allow any serum to drain away into the pleura. The lungs should be fully inflated as the chest is closed. The easiest way to be sure of this is to leave an under-water drainage tube passing obliquely through the wound but not stitched to it. After the dressings have been applied and the patient turned on to his back, positive pressure is applied through the anaesthetic face-piece to inflate the lungs and to drive out the last remaining air from the chest. The tube may then be pulled out quickly. If the passage through the tissues of the chest wall be oblique enough its sides will come together without allowing any air to re-enter the pleural cavity.

Post-operative treatment

After operation fluids and soft solids may be given from the beginning and the patient should be on an unrestricted diet after a week. He should usually be out of bed on the second post-operative day and walking by the third.

End-to-end anastomosis

The approach to tumours in the lower third of the oesophagus should be through the left pleural cavity after resection of the eighth or ninth rib. When the tumour causes obstruction by encircling the oesophagus it should be treated in the same way as a carcinoma in this situation except that there is of course no need to remove the spleen, pancreas and retroperitoneal cellular tissues. In one patient operated upon by the author the oesophagus was so dilated above the growth that after it had been transected it could be anastomosed end-to-end to the body of the stomach which had been mobilized and divided transversely. Not enough of these operations have been done for simple tumours to indicate whether repair should be by oesophago-gastrostomy or oesophago-enterostomy.

11. MALIGNANT TUMOURS OF THE OESOPHAGUS

Primary squamous-cell carcinoma

The commonest form of malignant disease of the oesophagus is a primary squamous-cell carcinoma, but secondary invasion from other organs occurs not infrequently, notably from the stomach below, from the lungs laterally by direct spread through the mediastinum, from the main bronchi at the level of the bifurcation of the trachea where the left main bronchus is in direct contact with the oesophagus, and from the thyroid gland in the neck. The three

Sites of occurrence

sites of election are at the upper end in the cervical region, the middle third at the level of the bifurcation of the trachea, and at the lower end, but the first of these is notably less common than the other two. These sites of occurrence are not always well defined, for a growth in the middle third may extend up to the upper third, and one at the lower end may reach as high as the bifurcation of the trachea. In addition to this, however, the oesophagus may undergo extensive malignant degeneration over its whole length or over the lower two-thirds. Apart from carcinoma there are reports of such rare tumours as sarcoma and rhabdomyosarcoma occurring in the oesophagus and it may be invaded by reticulum-cell sarcoma from the mediastinum.

Extensive malignant degeneration

12. CARCINOMA OF THE UPPER THIRD OF THE OESOPHAGUS

Incidence

Carcinoma of the upper third of the oesophagus is not common and occurs almost entirely in men, in contrast with the post-cricoid carcinoma which is almost confined to women. Early diagnosis is rare for it usually presents

either as a recurrent laryngeal paralysis, indicating spread of the tumour outside the oesophageal wall, or as malignant involvement of the glands at the root of the neck. The diagnosis is confirmed by x-ray and oesophagoscopy examinations.

Treatment

Successful surgical removal of these tumours is made difficult by late diagnosis, the proximity of the tumour to the trachea, to which it may soon become adherent, and by the confined space of the thoracic inlet in which it occurs. For these reasons it is often possible only to give radiation therapy and to insert a Souttar's tube to relieve dysphagia if there is enough normal oesophagus below the level of the cricoid cartilage to accommodate the expanded end of the tube. If the diagnosis is made early, surgical excision may be feasible. When the growth lies just below the cricoid it may be removed through a cervical approach and continuity restored by a skin tube, but it is unlikely that such a local operation will give a long survival rate. More often it will be necessary to do a combined thoracic and cervical approach and restore continuity later by a subcutaneous loop of jejunum according to the method used by Yudin for simple strictures.

Radiation therapy

Removal of growth

13. CARCINOMA OF THE MIDDLE THIRD OF THE OESOPHAGUS

Of primary squamous-cell carcinomas of the oesophagus, the one occurring in the middle third is reputed to be the most common. In a series of 277 patients reported by Raven (1948) carcinoma of the middle third was to the lower third in the proportion of 1.7 to 1. In 100 consecutive patients in the author's series, however, the lower-third lesion was just twice as common as that in the middle third.

(1) Morbid anatomy

The commonest form for the growth to take in this segment is a stenosing and infiltrating one which becomes adherent to the aorta, vena azygos and posterior wall of the trachea or main bronchi. The radiological appearance may be of a fairly smooth stenosis and the extension of the growth in the submucous layer may cause the top of the stenosis to be funnel-shaped and not ulcerated (Fig. 195). Only if the stenosis can be dilated may the ulceration of the mucosa be seen lower down. This annular type of lesion is usually short and when excised feels about the size and consistency of a bobbin.



Stenosis

FIG 195 —Constricting carcinoma of the middle third of oesophagus.

Less frequently a soft ulcerated growth occurs into the lumen, with correspondingly less spread into the mediastinum.

(2) Symptoms

Dysphagia

The earliest symptom is dysphagia, but it is important to remember that patients often pay no attention to this when it is mild. It may be intermittent, and indeed there is often a history of complete obstruction being spontaneously relieved, a feature which is accounted for by the temporary lodging of ill-masticated food which is subsequently displaced either upwards or downwards. Only in the later stages with spread outside the oesophagus may there

Pain

be pain going through to the back, respiratory symptoms due to bronchial involvement, and even tracheo-oesophageal fistula.

(3) Diagnosis

Oesophagoscopy

Bronchoscopy

The diagnosis should never depend on clinical and radiological examination alone. An oesophagoscopy should be done as a routine when a patient complains of dysphagia, for an early lesion may be found in this way which may easily be missed on the screen examination. Bronchoscopic examination should always be carried out if a carcinoma is found in the middle third of the oesophagus to see if the main bronchi are involved.

(4) Treatment

(a) Palliative measures

Palliative treatment by oesophagoscopy dilatation, intubation and sedatives are all that can be offered to those with late disease. Radiotherapy has much to offer and may cause the disappearance of the primary growth as seen by oesophagoscopy. It is also useful in relieving the pain of mediastinal involvement. Gastrostomy is contra-indicated.

(b) Operative treatment

Age of patient

If the growth can be diagnosed in its early stages it seems that radical surgical excision still offers the best hope of cure. Many ingenious operations have been devised from time to time, some involving multiple stages and some multiple incisions. It must be remembered, however, that these patients are usually over 60 years old. Numerous stages are a great burden and involve much suffering for these elderly people. A double approach which involves closing one incision, turning the patient into another position and making another incision upsets the cardio-respiratory balance and in any case is unnecessary. If a patient is to be treated surgically, there should be one operation during which the full field can be explored, the operability of the growth determined, the growth excised and continuity restored so that when the patient recovers from the anaesthetic the growth has been eradicated and swallowing is satisfactory.

14. ONE-STAGE EXCISION AND RECONSTRUCTION FOR CARCINOMA OF THE MIDDLE THIRD OF THE OESOPHAGUS

(1) Operative technique

Plan of *approach*

Under Pentothal Sodium anaesthesia oesophagoscopy should be performed and the oesophagus aspirated dry. An absorbent pack is left in the oesophagus just below the cricoid cartilage to prevent anything which may come up

through the stricture from reaching the pharynx and being aspirated into the lungs. A pharyngeal or an endotracheal tube may then be inserted so that inflation of the lungs can be controlled. A left-sided spinal anaesthetic of light Percaine is given or, if the patient's condition does not justify this, an intercostal nerve block may be used. An incision is made along the whole length of the eighth rib, crossing the costal margin in front and coming up over the erector spinae behind to the level of the fifth rib. The eighth rib is removed and the costal margin divided. The posterior ends of the seventh and sixth ribs are also removed and the sixth and seventh intercostal bundles divided. The chest is opened through the bed of the eighth rib, the lung is retracted forwards and the local lesion in the oesophagus inspected to make sure that it is removable.

Local anaesthetic may be injected into the superior mediastinum, the mediastinal pleura incised from the apex to the arch of the aorta and the oesophagus isolated above the growth. A light clamp is applied above the lesion to a part of the oesophagus which is to be removed. This clamp is left in position throughout the subsequent manipulation and effectively prevents infected blood or gastric contents from being displaced upwards into that part of the oesophagus which is to be opened for anastomosis. The arch of the aorta is then stripped in its posterior part and the vagus nerve divided below the recurrent laryngeal branch. The upper six left aortic intercostal arteries are divided between ligatures, the descending aorta displaced forwards and the corresponding right branches similarly divided (Fig. 196). Tapes may then be passed round the aorta to exert gentle traction whilst any oesophageal or bronchial vessels are ligated and cut. In this way the aorta is dissected off the oesophagus with safety. If an attempt is made to dissect the oesophagus off the aorta serious damage to the latter may occur by tearing a small branch off it in a position where bleeding may be most difficult to control. It is wise to abandon the local dissection at this stage for the freeing of the growth almost inevitably involves opening the right pleural cavity and it is better that this should not be open while the abdominal dissection is being done. The left phrenic nerve is crushed and the diaphragm split back from the anterior end of the incision into the oesophageal hiatus. The diaphragmatic vessels are clipped and then stitched. At the anterior end of the diaphragmatic incision

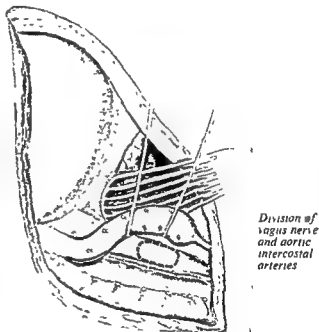


FIG. 196—Mobilization of the descending aorta by division of the upper intercostal and mediastinal vessels to expose a carcinoma of the middle third of the oesophagus.

Damage to aorta

Crushing of phrenic nerve

*Division of stomach**Separation of spleen**Division of oesophagus**Removal of growth**Removal of pancreas**Union of stomach and oesophagus**Penicillin*

the stitches are tied and then sutured to the external oblique muscle; at the posterior end they are left long and clipped outside the wound. By this means not only is the wound left widely open without the use of retractors, but the diaphragm is fixed and kinking of the inferior vena cava avoided. The filling of the heart is thus unimpaired. The vessels entering and leaving both curvatures of the stomach are divided from just below the cardia to within about 2 inches of the pylorus. The pyloric vessels themselves and the first few branches of the right gastro-epiploic vessels are left intact. The stomach is then divided between clamps just below the cardia and both ends are infolded and sutured. When the peritoneal reflection from the pyloric antrum on to the anterior aspect of the pancreas is divided the stomach may then be brought up into the left pleural cavity to see if it will reach to the superior mediastinum. When this has been confirmed the stomach may be replaced temporarily in the abdomen. The spleen is then separated from the diaphragm and splenic flexure and drawn forwards with the pancreas. The splenic vessels and neck of the pancreas are divided and the latter is sutured for haemostasis. The left gastric vessels are then divided and the body of the pancreas with spleen, vessels and left gastric and suprapancreatic glands dissected up to the cardia. The dissection is carried up in the mediastinum, leaving as much cellular tissue as possible adherent to the oesophagus.

Oesophageal vessels from the lower part of the descending aorta are divided and the lower margin of the growth is reached. The aorta is then drawn forwards and the growth-containing part of the oesophagus dissected out from the trachea and bronchi. It may be necessary to take the glands at the carina, part of the opposite mediastinal pleura and sometimes a segment of the arch of the azygos vein. This is easily done under direct vision from the left side when the aorta has been adequately mobilized. An anastomosis clamp is then applied to the oesophagus in the superior mediastinum 1 or 2 inches above the level chosen for section. The oesophagus is divided just above the first clamp which was applied at the beginning of the operation, and the growth, with the lower two-thirds of the oesophagus and its lymphatic bed, is removed in one piece. The removal of the pancreas is necessary if the suprapancreatic glands are to be taken, for it sometimes happens that the only metastasis from a middle-third growth is in this group of glands. The stomach is now drawn up behind the lung but outside the aortic arch and united to the end of the oesophagus, the latter being implanted into the anterior aspect of the fundus. The posterior wall of the oesophagus is first joined to the anterior wall of the stomach by interrupted sutures, the stomach is then opened and a continuous catgut stitch passed through all layers of both viscera to produce complete mucous membrane apposition. The anastomosis is completed by interrupted muscle sutures in front to bury the anastomosis in an "ink-well" of stomach. The stomach should then be lightly fixed to the parietal pleura to prevent longitudinal rotation, the diaphragm lightly closed around it and fixed to it by a few interrupted stitches. Penicillin powder is applied to the mediastinum and to the anastomosis. It is usually impossible to close the opposite pleura if it has been opened. It is better to make the opening larger, aspirate any blood which may have seeped down and aspirate the air by whatever means is used for the left pleura. This may be done by inflating the lungs during closure and then drawing off as much air as

possible by a pneumothorax apparatus when the wound is sealed, but this is not always quite complete for air tends to pocket or to come up from the abdomen. It is better to pass a drainage tube through the mediastinum at the lower end behind the stomach into the right pleura, make a lateral hole in the tube where it lies in the left pleura and bring it out obliquely through the chest wall. When this is connected to a water-seal all air is expelled from the abdomen and chest and when a skiagram shows this to be complete the tube can be withdrawn. This is usually after a few hours. It is important that

the tube should have a long oblique track through the wound and that it should be withdrawn smartly, for if the lateral hole is outside the chest while the end of the tube is in the pleura air will, of course, escape back into the chest. If both lungs are fully expanded there is less tendency for fluid to accumulate and more support for the anastomosis. It sometimes happens during inflation of the lungs that the stomach also becomes distended. When the operation is completed, therefore, and the patient turned on to his back, it is advisable to pass an oesophagoscope down to the anastomosis and insert a fine tube into the stomach. If this tube is left in until the patient has fully recovered consciousness it does no harm, the stomach can be aspirated to allow full lung expansion and respiratory efficiency is thereby increased. The tube should be removed on the next day as the presence of a foreign body is not conducive to the best healing at the anastomosis (Fig. 197)



Oblique track of tube

Distension of stomach

FIG. 197—Anastomosis of the oesophagus to the stomach in the left pleural cavity outside the aortic arch after radical removal of a carcinoma of the middle third of the oesophagus.

(2) Post-operative care

The patient should be nursed flat for 24 hours, but turned from side to side at intervals. Water may be given by mouth, but should be aspirated about every 2 hours during this time to prevent distension. After 24 hours the tube is removed from the stomach and only sips of fluid should be given until intestinal movements can be heard. Tea, coffee, water and fruit drinks may then be taken in moderation. The diet is increased from day to day so that the

Frequent aspiration of stomach

*Assistance
with
breathing and
coughing*

patient is taking a light mixed diet by the end of the week. The patient should have assistance with breathing and coughing throughout, and if his general condition is good he should be out of bed by the third day and walking about within the week.

(3) Complications

(a) Respiratory infection

*Inhalation of
secretions*

Respiratory infection is one of the most important complications of these extensive operations. The patients often have a chronic bronchial infection before operation, but apart from this the most important factor is the inhalation of septic secretions from the pharynx. These may be formed locally in the pharynx or may be regurgitated from the stomach or oesophagus. Only the most meticulous care during the patient's period of unconsciousness will avoid this and the steps already described have been elaborated to this end.

(b) Cardio-renal failure

Some patients succumb to what, for lack of more detailed knowledge, may be called cardio-renal failure.

(c) Faulty anastomosis

A leaking anastomosis is merely an indication of inaccurate technique, but a complete breaking down due to sloughing of the fundus of the stomach is caused by arteriosclerosis of the coeliac axis and its branches which ruins their elasticity and interferes with the blood supply of the fundus after section of the left gastric and gastro-epiploic trunks.

(d) Local sepsis, empyema and abscess formation

Penicillin

*Removal of
blood clot*

Local sepsis, empyema and subphrenic abscess may occur and should be treated along the lines laid down for these conditions. The liberal use of penicillin in the pleura and mediastinum, together with the insistence on complete expansion of the lungs at the end of the operation and the careful clearing out of any blood clot which may have collected at the apex of the pleura, has done much to reduce these complications.

(e) Gastric and intestinal stasis

Gastric and intestinal stasis may be troublesome or even fatal, but there is no very definite indication of what causes this in one patient but not in another. If the stomach distends it may embarrass respiration and in this event a fine catheter should be passed *via* the nose and aspiration maintained whilst the patient is fed intravenously.

(f) Pulmonary thrombosis and embolus

Other complications such as pulmonary thrombosis and embolus are common to all operations and do not need special description.

(g) Peptic ulceration

Stenosis

The only important late complication, apart from recurrence of growth, is peptic ulceration of the lower end of the oesophagus which may lead to stenosis. The stricture must be dilated by occasional passage of a bougie and the patient should learn to go to sleep sitting up in order to diminish the reflux of acid during the night.

15. CARCINOMA OF THE LOWER THIRD OF THE OESOPHAGUS

In the author's experience the lower third is the commonest site for primary oesophageal carcinoma, the squamous growth being twice as common as that in the middle third. In addition to this, however, the lower end of the oesophagus is frequently involved by a carcinoma extending up from the stomach. The latter is an adenocarcinoma and maintains its histological features in the oesophagus. A primary squamous growth may involve the stomach, and it is necessary, therefore, to consider these together as malignant disease of the gastro-oesophageal junction. Their distinction from the surgical point of view merely leads to a difference in the level at which the oesophagus is divided in the mediastinum, for their lymphatic drainage is identical and the general principles of treatment are therefore the same.

(1) Morbid anatomy

If the tumour grows into the lumen of the oesophagus it takes on a sausage-like shape unless it starts very near the cardia and prolapses into the stomach, when it may become spherical. The importance of the shape is that what appears radiologically and oesophagoscopically to be a very extensive growth may be attached to the oesophageal wall by a relatively small pedicle. It is unfortunate that these polypoid tumours do not cause very distressing symptoms, so that by the time the diagnosis is made lymphatic involvement may be widespread even when local infiltration is minimal. An annular type of growth is not common in the lower third, but sometimes the spread into the mediastinum is much more extensive than the local ulceration would suggest. Most commonly there is heaping up of tissue in the lumen, ulceration, and infiltration in and through the oesophageal wall in proportion. When a gastric growth extends into the oesophagus, ulceration usually keeps pace with proliferation, but it is not uncommon for the growth to extend upwards in the extra-mucosal planes leaving the mucosa smooth, in which case a smooth funnel-shaped stenosis is formed which may be mistaken for cardiospasm. As the growth extends into the mediastinum it may involve the diaphragm, aorta, pleurae and lungs or the pericardium.

(2) Lymphatic drainage of the lower third of the oesophagus

The spread to lymphatic glands is primarily into those of the posterior mediastinum. These occur on all aspects of the oesophagus and extend into the lateral pulmonary ligaments. The main flow is downwards into the glands around the cardia and along the left gastric group to those around the coeliac axis. At the cardia, cells may be diverted into the suprapancreatic glands and malignant invasion of glands above the tail of the pancreas has been seen even when the primary growth has not transgressed the confines of the oesophagus. Although the main direction is downwards there is also a lymphatic spread upwards to the posterior mediastinal, tracheo-bronchial and cervical glands. The cervical glands may be detected on clinical examination and so forestall surgery, but the others may be found only when the chest is opened. They may, of course, easily be missed even by direct examination of the mediastinum. The radical excision of a carcinoma of the lower third of the oesophagus therefore involves removing the lower end of the oesophagus

Adrenaline is added to the above solution immediately before use in the proportion of 10 minims of 0.001 per cent adrenaline to 250 cubic centimetres of solution.

(4) Operation

The full thoraco-abdominal incision extends from the outer border of the *Incision* left rectus abdominus, or if necessary through the outer part of this muscle, and sweeps backwards crossing the costal margin at the tip of the ninth cartilage. It follows the ninth rib back to the outer border of the erector spinae muscle where it curves upwards for 2 inches. After the skin incision has been made the muscles are divided with the diathermy. The posterior end of the eighth rib and the whole of the ninth rib are removed. At the anterior end the costal cartilage is dissected away from the muscles with the diathermy knife. The pleura and peritoneum are opened. The diaphragm is split back towards the oesophageal hiatus from the anterior end of the pleural incision. As the vessels in the diaphragm are caught they are stitched. It is convenient to use these *Use of stitches for retraction* stitches as retractors, the anterior ones being sewn to the external oblique muscle on the edges of the incision, and the posterior ones being left long and anchored on the surface by artery forceps. The fixation of the diaphragm in this way not only makes retractors unnecessary, but allows proper action of the right dome of the diaphragm and prevents the pull of this from kinking the inferior vena cava and obstructing the blood return to the heart. When this incision has been completed there is no need for retractors for the edges fall apart and there is ample room to do both the abdominal and thoracic dissections. The mediastinal pleura is incised at the level at which the oesophagus is to be divided. The vagus nerves are divided and the oesophagus is drawn out from its bed. A thin anastomosis clamp is then applied to the oesophagus just below the point chosen for section and firmly closed so that there is no possibility of infected or irritating fluids escaping upwards during the mobilization of the stomach. The growth is dissected away from the surrounding structures, portions of both pleurae being removed if necessary. *Incision in mediastinal pleura*

The spleen is drawn forwards and the outer layer of the lienorenal ligament divided. The division of this layer of peritoneum is carried up over the diaphragm nearly to the hiatus. Traction on the spleen and a combination of sharp and blunt dissection then displaces the whole stomach bed forwards. If the separation is easy the adrenal gland is left behind, but if there is fixation the adrenal may be taken forwards with the spleen, splenic vessels and pancreas. The left crus of the diaphragm where it is fanning out is cleaned and divided into the hiatus after ligation and division of the inferior phrenic vessels. The sustentaculum lienis is divided and the lesser sac opened below the spleen. As the pancreas is drawn forwards the peritoneal reflection from the lower border is divided until the inferior mesenteric vein is reached. The fold of peritoneum between the pyloric antrum and the pancreas is divided and the duodenum freed as far as the pancreaticoduodenal vessels which lie on its posterior surface. With the mass of tissue still displaced forwards the splenic vein is isolated where the inferior mesenteric vein joins it and it is divided between ligatures so as to leave the tributary from the large intestine intact. *Division of left crus of diaphragm*
Isolation and division of

*Division of
splenic artery*

it crosses to join either the splenic or portal vein. The pancreas is divided with the diathermy knife and a few bleeding vessels in its substance are secured with a running catgut stitch. The stomach is still held to the posterior abdominal wall by the left gastric artery and some cellular and lymphatic tissue. The vessel is cleaned and divided at its origin and the surrounding lymphatic and nerve tissue, which often contains small veins, is ligated and divided. Only the left half of the great omentum is removed with the *fundus* of the stomach. The dissection along the greater curvature of the stomach is not taken as far as the pylorus, but 4 or 5 of the branches of the right gastro-epiploic vessels are left intact. The pyloric vessels are not divided but a point is chosen on the lesser curvature of the stomach where they anastomose with the left gastric vessels. The lesser omentum is divided here and the adjacent part of the curvature cleared of vessels. Clamps are then applied obliquely from this position across the stomach to the point on the greater curvature which has been cleared of great omentum. The stomach is divided and closed, the oesophagus clamped and divided, and the mass of tissue containing the growth is removed. The remaining part of the stomach is swung up into the mediastinum and joined to the cut end of the oesophagus. The posterior wall of the oesophagus is first united to the front of the stomach just below the top of the latter by a series of interrupted catgut-stitches. The stomach is opened and a continuous stitch passed round the whole circumference to include all layers. Interrupted muscle stitches in front then cover the anastomosis with peritoneum. The stomach is stitched to the pleura and

*Removal of
growth*

to the edge of the cut diaphragm and the latter is loosely closed around it

(5) Post-operative treatment

The post-operative treatment is the same as that already described for resection of the middle third of the oesophagus.



FIG. 198 —Palliative oesophago-jejunostomy above an irremovable carcinoma of the cardia

16. OESOPHAGO-JEJUNOSTOMY WITH RESECTION FOR CARCINOMA OF THE CARDIA

If there is evidence of arteriosclerosis of the *coeliac artery* and its branches and if the surgeon feels, as some do, that oesophago-gastrostomy is associated with too high a proportion of dyspeptic symptoms and even

ulceration with stenosis, it is reasonable to remove the whole of the stomach and close the duodenum. A loop of jejunum is then fashioned according to the method of Roux (already described for the treatment of peptic ulcer of the oesophagus by excision) and this is anastomosed to the lower end of the oesophagus. *Method of Roux*

17. PALLIATIVE OESOPHAGO-JEJUNOSTOMY FOR IRREMOVABLE CARCINOMA OF THE LOWER THIRD OF THE OESOPHAGUS

If the thoraco-abdominal incision has been made and the chest and abdomen widely opened, it may only then become apparent that the growth is inoperable. If this is due to secondary deposits at a distance, for example in the liver, it may still be worth while to resect the primary growth and restore continuity so that the patient can swallow. If, however, the growth is irre- *Resection of primary growth* movable by virtue of local fixation relief may be given by bringing up a loop of intestine on the Roux principle through the transverse mesocolon, behind the stomach and pancreas, through the diaphragm and into the mediastinum above the growth. A side-to-side anastomosis may then be made to the oesophagus and an effective by-pass established for swallowing. This operation may allow the patient to spend his last months in comparative comfort, swallowing normally (Fig. 198).

REFERENCES

Raven, R. W. (1948). *Brit. J. Surg*, 36, 70.
[References to other titles are given under Oesophagus in the Index Volume. The subject is also dealt with under the heading of Oesophagus Diseases in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 287.]

OMENTUM

By A. J. COKKINIS, F.R.C.S.
SURGEON, WEMBLEY HOSPITAL

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1. EPILOITIS AND OMENTAL ABSCESS

Pathology

fecting property appears to be the main function of the human omentum. Only rarely does the omentum itself yield to the infection and become involved in a spreading suppuration of its cellular tissue (epiploitis), perhaps ending in an omental abscess.

Treatment

A correct diagnosis is unlikely before the abdomen is explored. If an abscess is found it is opened and drained. Necrosed omentum is excised but adherent omentum should be left alone.

2. TORSION OF THE OMENTUM

(1) Pathology

Torsion, a lesion of real surgical interest, may involve the whole omentum or part of it, and may be complete (strangulation) or partial (congestion). About 200 cases have been reported (Wilson, 1945). Always clockwise, the torsion is either primary (idiopathic) or secondary. *Varieties*

(a) Primary torsion

Primary torsion is less common and in about half the number of cases involves the entire omentum. Predisposing factors are (i) eccentric weight distribution—for example, fat deposits at its fringe (Anton, Jennings and Spiegel, 1945); (ii) extra room in the abdomen—for example, an obese person who has lost weight; (iii) a long and pedicled omentum (congenital or pulled down by fat). Activating causes are external violence producing an axial twist, powerful peristalsis squeezing a "ball of omental fat" against the abdominal wall, or violent contraction of the wall itself.

(b) Secondary torsion

Secondary torsion is seen mostly in connexion with right inguinal hernia. Taxis is the common activating cause. The twist starts in the sac, but may extend into the abdomen and involve the right half of the omentum. Other predisposing causes of secondary torsion are adhesions of omentum to an inflamed focus, and omental tumours or cysts.

The morbid changes are congestion and oedema. If the twist is complete (often several turns), the omentum becomes infarcted and finally gangrenous. Blood-stained serum is extravasated into the omentum, the peritoneum and the hernial sac (if present). Should the strangulation stop short of gangrene, the omentum may atrophy or become revascularized through adhesions. *Morbid anatomy*

(2) Diagnosis

Never easy, this is unlikely to be made in primary cases. The presence of a tender and tense right inguinal hernia (especially after taxis) is of obvious help. The usual mistakes are appendicitis with a lump, twisted ovarian cyst or acute cholecystitis.

Suggestive features are sudden pain in the lower right abdomen in a male aged 30–50 years, nausea and vomiting, mild fever, and a tender mobile and ill-defined mass. When this can be felt to extend from a hernia towards the umbilicus the diagnosis should not be difficult. But in idiopathic cases the mass will almost certainly be mistaken for a localizing appendicular peritonitis, except when it lies higher in the abdomen in the gall-bladder region.

(3) Treatment

Laparotomy is essential whether a diagnosis is reached or not. Blood-stained fluid will be present. The finger will at once come upon the mass of twisted omentum, which feels very like a sponge. It must not be mistaken for a plastic mass surrounding an inflamed appendix or gall-bladder. The mass is followed up to its pedicle and removed, by tying off well on its proximal side. If the whole omentum is involved it should be removed in its entirety. *Operation*

3. TUMOURS AND CYSTS

(1) Tumours

(a) Primary neoplasms

The only primary neoplasm of importance is fibrosarcoma. It grows slowly and is found when exploring a mass of doubtful origin. Intestinal obstruction occurs only when the growth is very large (Maingot, 1940). Treatment consists in excising the entire omentum and any adherent intestine together with a wedge of its mesentery.

(b) Secondary neoplasms

Secondary carcinoma is of course common: the primary is to be found in the breast, stomach, colon or ovary. It is associated with ascites and nodular tumours in the pelvis.

(2) Cysts

The commonest cyst is a cavernous lymphangioma. It tends to be multiple and to resemble a bunch of grapes. The only safe treatment is to resect the whole omentum. Rarer cysts are hydatids and dermoids.

4. THE OMENTUM IN ABDOMINAL LESIONS

The part played by the omentum in the morbid anatomy of abdominal adhesions, obstruction and hernia is known to every general surgeon. In this place it would be superfluous to do more than refresh his memory on some salient points.

(1) Omental adhesions

The surface and fringes of the omentum adhere readily to inflamed foci and denuded areas alike. Normally this happens in the abdomen only, but with a ptosed stomach the omentum may drop into, and become adherent in, the pelvis.

Diffuse adhesions

In chronic peritoneal infections the omentum becomes extensively adherent to viscera and parietes, or it may be "rolled up" into a thick hypertrophied mass stretching across the abdomen.

(2) Intestinal obstruction

Local adhesions

A local adhesion may stretch and organize into a band, which produces mechanical obstruction by kinking or snaring a loop of intestine. Owing to the mobility of the greater curve of the stomach actual strangulation of gut is unlikely, unless the omental band has a fixed point of attachment proximally as well as distally.

5. THE OMENTUM AND HERNIA

Most hernias contain omentum (epiplocele). Its length, weight, lobulated fringes and "creeping tendency" cause it to engage in hernial orifices. It adheres to both sac and other contents. It is the most common single content of femoral hernia. In inguinal and umbilical hernia it is usually found with intestine. In conditions of strangulation, omentum is less liable to necrosis than is the intestine, because it can acquire a collateral circulation through its adhesions to the sac.

An omental hernia shows itself as a spongy, lobulated mass. It has a weak impulse, is dull on percussion and is usually irreducible. *Diagnosis*

Dangers of omentum in a hernia

The following difficulties and dangers are associated with omentum in a hernia.

(a) Omental sac

The free margin of the omentum may adhere to the posterior edge of the aperture and the omentum above may be invaginated into the hernial sac to form a second "omental sac". This may contain a knuckle of gut which would obviously be endangered during operation.

(b) Irreducibility

Omentum tends to make a hernia irreducible because it adheres to the sac. In time the omentum becomes hypertrophied and this may lead to incarceration of the hernia.

(c) Loculation

Omentum is a predisposing cause of loculation. By adhering densely to the sac below the neck, the omentum may force the wall above to bulge out into one or more loculi. There may be a gangrenous fringe of omentum in a deep loculus, but the main sac is empty or has healthy contents.

(d) Adhesions and torsion

In large umbilical and incisional hernias extensive omental adhesions and associated loculation often present great difficulties at operation. Torsion of the omentum in a hernia has been dealt with already. Omentum fixed in a hernial orifice constitutes a band across the peritoneal cavity which may cause intestinal obstruction.

6. USES OF THE OMENTUM IN OPERATIVE SURGERY

Nothing is more characteristic of the resourcefulness of modern surgery than the uses it makes of the omentum. These uses are based on four discoveries, one old and three new. The old one is that the omentum will adhere to almost anything. The new ones are: (i) that it can act as a source of new blood supply to an ischaemic structure; (ii) that free portions of it survive when transplanted; and (iii) that omental grafts, free or attached, stimulate and increase the vascularity of the bed to which they are transplanted. *Basis of omental surgery*

(1) Uses of the attached omentum

(a) In abdominal operations

The attached omentum has been used for very many years as a general protector in abdominal surgery. It is still regularly employed (i) to cover duodenal stumps, sutured peptic ulcers and raw areas after cholecystectomy; (ii) round enterostomy tubes, intestinal anastomoses and gut of doubtful vitality; (iii) under abdominal incisions when parietal peritoneum is deficient; (iv) to check bleeding from tears of the liver and spleen. *Omental "cover" in abdominal surgery*

Using the attached omentum is not quite free from danger. The following accidents are possible: *Dangers of using attached omentum*

(i) Traction on the stomach and transverse colon may interfere with their function, especially if the omentum is pulled into the pelvis; (ii) volvulus of

the small gut may occur round an omenta pedicle; (iii) the omentum may act as an obstructing band.

These dangers can be avoided by using free omental grafts, which serve the above purposes just as well (*see p. 359*).

(b) *In portal obstruction*

*Omentopexy
for cirrhosis
of the liver*

The operation of omentopexy for cirrhosis of the liver was first performed by Rutherford Morison in 1896, but opinions still differ on whether it is justifiable or not. The mortality within 2 weeks of the operation is about 40 per cent (Cates, 1943). Only early cases of hypertrophic cirrhosis, without jaundice or hepatic deficiency and preferably without ascites, are likely to benefit (Bodenheimer, 1942). (*See Liver-Cirrhosis, Vol. 5, p. 437.*)

Technique

The technique of the operation varies with the anaesthetic. Under local anaesthesia the omentum is brought out through a small incision and sutured to the abdominal muscles (lower mortality but less likely to benefit). With general anaesthesia the risk is greater but more can be done to get a collateral blood supply by roughening the spleen and the diaphragmatic surface of the liver.

After-treatment

Paracentesis may be needed after operation, probably several times, and may help by producing adhesions which become vascularized. Though the blood volume is low in cirrhosis (because of hypoproteinaemia) attempts to raise it by intravenous fluid are dangerous, because anasarca and pulmonary oedema are likely consequences (due to disturbed osmotic equilibrium).

(c) *In cardiac ischaemia*

The operation of cardio-omentopexy is described and illustrated in the section on Angina Pectoris (*see Vol. 1, p. 254, and Figs. 94-96 in that section*).

(d) *In renal ischaemia (arterial hypertension)*

Because renal ischaemia is believed to be a factor in the aetiology of essential hypertension, improvement might be expected if the blood supply of the renal cortex could be increased by collateral routes.

*Technique of
nephro-
omentopexy*

The operation of nephro-omentopexy has been devised with this object in view. An anterior transperitoneal approach to the right kidney is made through a pararectal incision. The caecum and right colon are mobilized by an incision lateral to them and pushed inward (Fig. 199 (a) and (b)). The kidney is freed and delivered into the abdomen. Its capsule is split along the convex border and both anterior and posterior halves are excised to near the renal pelvis, leaving a good fringe on the posterior half. A small piece of renal cortex is excised for biopsy, the wound being closed with a mattress suture. The kidney is now seared lightly with the cautery. The next step is to divide the omentum along its middle, avoiding the main vessels. Its right half is picked up by three sutures, the other ends of which are passed through corresponding parts of the posterior fringe of the renal capsule (Fig. 199 (c)). The right half of the omentum is now swung up to the kidney and as the sutures are tied the two structures will make complete contact. Lastly, the kidney is pushed back into the loin and the incision in the external paracolic sulcus is closed as far as the point at which the omentum leaves the peritoneal cavity.

Although temporary improvement has been reported by several writers *Results* (Bruger and Carter, 1941; Cottett and Rudler, 1945) it is too early to express an opinion on the long-term results of this procedure.

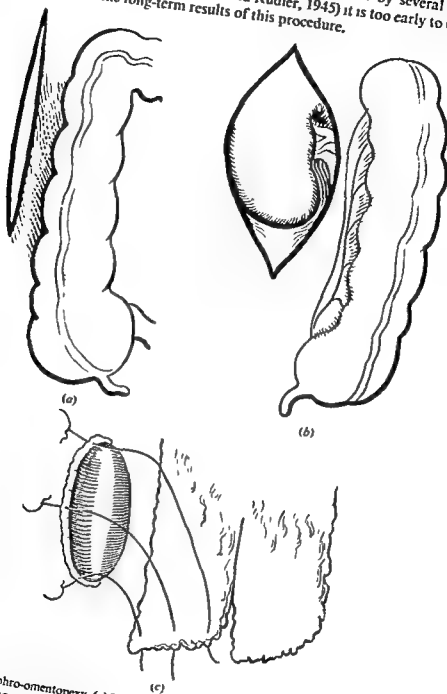


FIG. 199.—Nephro-omentopexy. (a) First stage, showing incision of posterior peritoneum; (b) second stage, showing exposure of kidney; (c) third stage, showing method of suturing omentum to fringe of renal capsule.

(2) Uses of the free omentum

The use of the free omental grafts is now an established surgical advance. They are known to survive, to become rapidly vascularized and to provide a

smooth endothelial covering for raw or rough surfaces. Certain rules must be observed if uniform success is to be obtained. We shall limit our discussion to a consideration of these rules, to details of technique and to indications for the procedure.

(a) *Indications in abdominal surgery*

*Omental
grafts in
abdominal
surgery*

(i) To cover denuded visceral and other peritoneal surfaces, with the object of preventing adhesions and adhesive intestinal obstruction. It need not be stressed that this type of obstruction is recurrent and that the more often adhesions are broken down the more obstinately they re-form. The object of the omental graft is to break this vicious circle by re-peritonealizing all denuded surfaces.

(ii) To check bleeding from the liver and other solid organs (Graham, 1934).

(iii) For purposes for which attached omentum has been used in the past, such as reinforcing the repair of perforated peptic ulcers with friable edges (Kirkpatrick, 1942), and wrapping round duodenal stumps and intestinal anastomoses.

(b) *Indications outside the abdomen*

*Free omental
grafts outside
the abdomen*

(i) To stimulate the healing of wounds of various types (Mandl and Rabinovici, 1946).

(ii) To close a cerebrospinal-fluid fistula (Mandl, 1944).

(iii) In certain thoracic procedures (Thompson and Pollock, 1945).

(c) *Survival of the grafts*

The following rules must be observed if the grafts are to survive, to resist infection and to succeed in providing a complete covering (Freeman, 1916; Mann, 1921).

*Rules for
omental
grafting*

(i) They must be taken from the free border of the omentum (to avoid large vessels).

(ii) The utmost gentleness must be exercised in handling them, rubber-covered instruments and the finest intestinal needles and catgut being used. The grafts should be kept in warm saline solution whilst awaiting transplantation.

(iii) The ligated pedicles must not be puckered, and the raw edges left behind must be re-peritonealized.

(iv) The graft must be large enough to overlap the defect everywhere, but not larger. Careful measurements, therefore, are necessary.

(v) The graft is sutured to its bed without tension, and its edges are turned in to avoid any exposure of the cut surface and subsequent adhesions.

(d) *Technique*

*Example of
operative
technique*

A suitable case for omental grafting would be a chronic cholecystitis, with ...
mass. Large denuded and rough areas are left on the liver, duodenum ...
colon. After bleeding has been checked and the raw areas have been measured,
grafts are cut from the thin free border of the omentum with fine scissors,
using rubber-covered forceps to hold the omentum. Each graft is large
enough to overlap the corresponding defect by $\frac{1}{2}$ centimetre. The grafts are
placed in warm saline solution.

The next step is to re-peritonealize the cut edge of the omentum, which otherwise would be bound to adhere somewhere. As the grafts are cut omental vessels will need to be picked up and ligated. This is done with No. 00

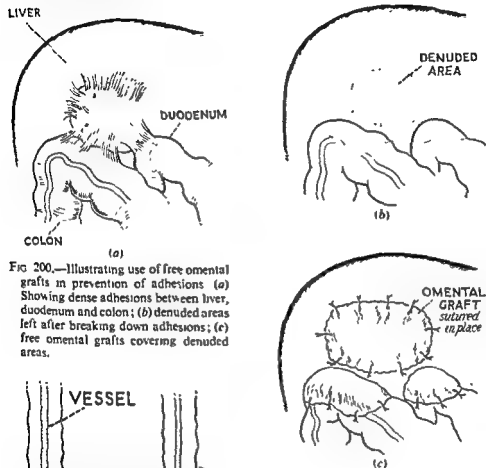


FIG. 200.—Illustrating use of free omental grafts in prevention of adhesions (a) Showing dense adhesions between liver, duodenum and colon; (b) denuded areas left after breaking down adhesions; (c) free omental grafts covering denuded areas.

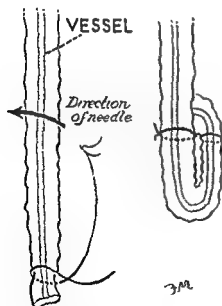


FIG. 201.—Diagram of method of folding omentum to cover raw edge after removal of graft.

catgut and the ends of the ligatures are left long (Fig. 201). Each end of each ligature is then threaded to a round-bodied needle. The blunt end of this needle is passed through the intact omentum $\frac{1}{2}$ inch higher up and $\frac{1}{2}$ inch from its fellow. As the two ends are tied the cut surface is made to roll under and becomes covered (McGehee and Tendler, 1942).

The grafts are then sutured in position with No. 0000 catgut on intestinal needles. Interrupted sutures are placed 2 centimetres apart, and as they are tied the edge of the graft is tucked under. No tension whatever is permissible.

REFERENCES

- Anton, J. I., Jennings, J. E., and Spiegel, M. B. (1945). *Amer. J. Surg.*, **68**, 303.
Bodenheimer, J. M. (1942). *Amer. J. Surg.*, **57**, 151.
Bruger, M., and Carter, R. F. (1941). *Ann. Surg.*, **113**, 381.
Cates, H. B. (1943). *Arch. intern. Med.*, **71**, 183.
Cottett, J., and Rudler, J. C. (1945). *Bull. Soc. méd. Hôp. Paris*, **61**, 204.
Freeman, L. (1916). *Ann. Surg.*, **63**, 83.
Graham, H. F. (1934). *Ann. Surg.*, **100**, 960.
Kirkpatrick, G. J. A. (1942). *Canad. med. Ass. J.*, **46**, 168.
McGehee, J. L., and Tendler, M. J. (1942). *Surg. Gynec. Obstet.*, **74**, 1046.
Maingot, R. (1940). *Abdominal Operations*. London; Appleton-Century.
Mandl, F. (1944). *J. Int. Coll. Surg.*, **7**, 219.
— and Rabinovici, N. (1946). *J. Int. Coll. Surg.*, **9**, 525.
Mann, F. C. (1921). *Surg. Clin. N. Amer.*, **1**, 1465.
Thompson, S. A., and Pollock, B. (1945). *Amer. J. Surg.*, **70**, 227.
Wilson, W. E. (1945). *Proc. R. Soc. Med.*, **38**, 185.

[References to other titles are given under Omentum in the Index Volume.]

OPTIC NERVE

By W. J. B. RIDDELL, M.D., F.R.F.P.S.

PROFESSOR OF OPHTHALMOLOGY, UNIVERSITY OF GLASGOW; VISITING
OPHTHALMIC SURGEON, WESTERN INFIRMARY, GLASGOW

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(2) Naffziger's operation - - - - -	368

1. SCOPE

249.] The optic nerve is in a well-protected position anatomically, and direct injury is relatively rare; it is frequently damaged in head injuries, particularly in fractures of the skull involving the middle fossa.

The common nervous diseases which affect the optic nerve do not come directly within the scope of the surgeon. The two outstanding categories which may require surgical intervention are tumours of the nerve itself and vascular lesions which interfere with its function. The signs and symptoms affecting the optic nerve secondarily to the presence of an intracranial tumour are beyond the scope of this article.

*Tumours of
optic nerve
and vascular
lesions*

2. ANATOMICAL RELATIONS

(i) *Optic nerve*.—Within the orbit, the nerve follows a complex, elongated, S-shaped course from the back of the globe to the optic foramen. There are two main curves, a posterior one with its convexity outwards and an anterior one with its convexity downwards (Fig. 202). It is protected from minor trauma by the bony walls of the orbit, and it frequently escapes damage in severe cases of orbital cellulitis because of the protection afforded by the optic nerve sheath. It has been stated, in such of the latter cases in which optic atrophy develops, that the damage may arise from mechanical stretching of the nerve and not from direct toxic action upon the structure itself. In cases of malignant exophthalmos in which there is no orbital inflammation, this stretching mechanism may well be the cause of optic atrophy.

*Mechanical
stretching of
nerve*

(ii) *Optic disc*.—The optic nerve is seen with the ophthalmoscope as the optic disc or papilla at the back of the globe. In this situation, the nerve fibres have no myelin sheaths, and there is little supporting tissue. Immediately outside the globe, the myelin sheaths are present. There is more supporting tissue between the nerve fibres, and as a consequence the diameter of the nerve is doubled. This is usually stated to be 1.5 millimetres at the disc and 3 millimetres as the nerve emerges from the globe. The lamina cribrosa is a

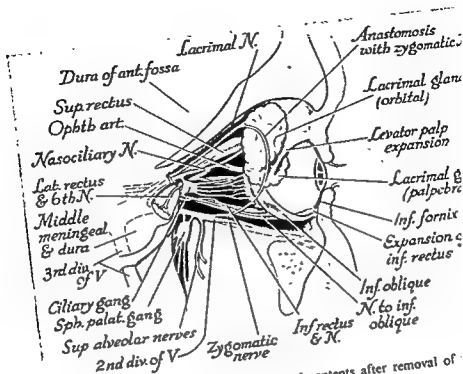


FIG. 202.—Diagrammatic view of the orbital contents after removal of bony wall.

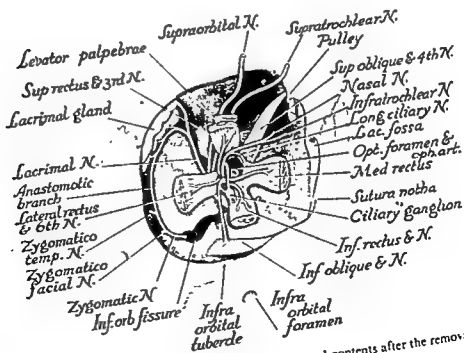


FIG. 203.—Diagrammatic anterior view of the orbital contents after the removal of the eyelids, globe and orbital fat.

series of transverse membranes through which the nerve fibres pass on their way from the retina to the optic nerve proper outside the globe. The inner layers consist of glial tissue, but the outer ones are made up of a mixture of glial and connective tissue.

(iii) *Ophthalmic artery*.—The ophthalmic artery arises from the internal carotid artery as the latter emerges from the cavernous sinus. It is medial to the anterior clinoid process and deep to the optic nerve. The artery enters the orbit through the optic foramen or canal in the dural sheath of the optic nerve. It passes forwards within the muscle cone, with the lateral rectus and the ciliary ganglion on one side and the optic nerve on the other. The central retinal artery comes off the main vessel shortly after it enters the orbit, and lies below the nerve until the point of entry within the nerve; this is usually 10–15 millimetres behind the globe.

(iv) *Posterior ciliary vessels*.—The posterior ciliary vessels arise from 2 main trunks from the ophthalmic artery, and divide into 10–20 smaller branches, forming the short posterior ciliary vessels which supply the choroid. Two branches which enter the globe, one on each side of the optic nerve, are called the long posterior ciliary vessels; they ultimately supply the ciliary body and the iris along with the anterior ciliary arteries. The ciliary vessels may be torn in avulsion of the optic nerve at its entrance to the globe.

(v) *Attachment to muscle cone*.—Within the orbit the muscles are separated from the nerve by the orbital fat, but at the apex the nerve comes into close proximity with the origin of the extra-ocular muscle cone (Fig. 203). It is particularly adherent to the superior and internal recti, and this attachment is the origin of the pain on movement of the globe which may be found in lesions of the optic nerve, such as retrobulbar neuritis. Origin of pain

(vi) *Optic foramen or canal*.—In the optic foramen or canal, the nerve is closely surrounded by its covering of dura, arachnoid and pia. These structures are all firmly bound together, to themselves and also to the periosteum without and the nerve within. This forms a point of fixation for the nerve and it cannot slide in and out of the cranial cavity.

On the medial aspect, the nerve is separated from the sphenoidal air sinus and the posterior ethmoidal air cells by an extremely thin layer of bone. It is this relationship which is used to explain the occurrence of retrobulbar neuritis in disease of the nasal sinuses. Retrobulbar neuritis

(vii) *Intracranial portion of the optic nerve*.—The intracranial portion of the nerve is about 1 centimetre in length, measured from the posterior end of the optic canal to the anterior aspect of the optic chiasma. It becomes somewhat flattened and loses its dural covering. The pituitary body and the anterior portion of the cavernous sinus lie below, and the terminal portion of the internal carotid artery lies laterally.

The whole length of the nerve is about 5 centimetres or 2 inches, the orbital portion being 3·5 centimetres, that in the optic canal 0·5 centimetre and the intracranial portion 1 centimetre.

3. HEAD INJURIES

Head injuries may affect the nerve by pressure from extravasated intracranial haemorrhage, through fracture of the optic canal or by direct injury to the nerve as in battle and air-raid casualties. Except in severe crush injuries, the Haemorrhage into optic nerve sheath

OPTIC NERVE

Orbital portion usually escapes, although there may be haemorrhage into the nerve sheath. This may arise through the blood tracking along the sheath from within the skull, or from damage to the capillary vessels crossing the intervaginal space.

4. LOCAL INJURIES

In civilian life, injuries to the orbital portion of the optic nerve fall into two main classes. The first are penetrating wounds of the orbit, arising from relatively high-velocity missiles, such as shot-gun pellets. In the second class are those caused by splinters of wood, lead pencils, umbrella-spokes or similar objects.

(1) Avulsion of optic nerve

The optic nerve may be partially or completely torn from the globe it Such an injury may arise from diverse activities, as in a game like water-polo, in which an opponent may violently displace the eye upwards and inwards with his foot or finger, or on a farm where a friendly cow may raise her untethered head and cause avulsion of the optic nerve with her horn. I have known the optic nerve to be damaged in the course of an operation upon the nasal accessory air sinuses. Fibres of the optic nerve degenerate centrally and peripherally after injury. In this respect, the reaction is similar to damaged brain tissue, and there is no attempt at regeneration.

(2) Fracture of facial bones

Fracture of the facial bones in motor-car injuries may lead to extensive haemorrhage within the orbit and damage to the optic nerve. Many such cases are best handled by collaboration with a facio-maxillary surgeon. It is of importance that fractures involving displacement of the orbital margins should be corrected as early as possible before callus formation interferes with anatomical replacement.

5. GENERAL DISEASES

There are no general diseases affecting the optic nerve which are amenable to surgical intervention, treatment depends entirely upon attacking the primary cause.

(i) *Orbital cellulitis*.—Orbital cellulitis arises frequently from infections of the accessory nasal air sinuses, particularly the ethmoid cells. The increase in orbital pressure leads to exophthalmos and to stretching of the optic nerve. In many cases, this is due primarily to oedema and not to pus formation. Many observers believe that the improved drainage obtained by a well-designed incision has saved the sight of the eye, although no pus may be found. The incision must on no account be made along the line of the upper orbital margin. Division of the levator palpebrae superioris and subsequent scar-tissue formation may give rise to a gross deformity which is excessively difficult to rectify by subsequent operations. The incision should follow the natural lines of the skin folds, and a curved incision at the upper inner angle frequently attains this object. In order to achieve effective release of pressure within the orbit, the incision must penetrate the septum orbitale. If only a shallow incision is made, no effective drainage will result.

Types of
injury

Degeneration
of nerve fibres

Callus
formation

vision

(ii) *Haemorrhage*.—Haemorrhage into the optic nerve sheath may arise in cases of subarachnoid haemorrhage. It has been shown by Ballantyne (1943) that there is not necessarily any direct connexion between lesions arising within the skull, in the orbit and within the globe, except a generalized vascular degeneration. The hypothesis that the haemorrhage originates in one situation and effuses or gravitates into the others is not universally true. Such multiple haemorrhages may be explained by a stasis in all the venous channels which drain the tissues of the eye and the contents of the orbit. *Vascular degeneration*

6. NEOPLASMS

The optic nerve is a part of the brain, and consequently the tumours of the optic nerve fall into two main classes: gliomas of the nerve, which correspond to the glioma of brain tissue, and meningiomas or endotheliomas of the nerve coverings.

Glioma of the optic nerve occurs most frequently about 10–15 millimetres behind the globe. The growth spreads towards the brain, because the anterior portion of the nerve contains a greater proportion of non-neural tissue which these tumours do not invade. The central retinal artery and the denser septa tend to form an inadequate protective barrier. Clinically this results in relatively little limitation of movement of the globe compared with that caused by meningeal tumours. A glioma of the optic nerve forms a localized thickening, and develops as an egg-shaped mass occupying the orbit. The dura remains intact. *Glioma of optic nerve*

Meningioma of the optic nerve grows on the dura and leads to compression of the nerve itself. These growths tend to be conditioned by the shape of the muscle cone, and may even form a cup shape at the back of the globe. Gross limitation of ocular movement may occur at an earlier stage than in the gliomas. *Meningioma of sheath*

Neither of these tumours tends to spread into the eyeball itself, although exceptional cases are on record. They both cause protrusion of the globe, and are relatively slow-growing. Any sudden increase in the exophthalmos is usually due to haemorrhage within the growth. Obviously, vision will be lost more rapidly in glioma than in meningioma. They tend to occur in different age-groups—the gliomas before the age of 20, and the meningiomas between the ages of 20 and 40 years. They are rare in older patients. Malignancy is low and metastases are very rare. Other neoplasms of the optic nerve are great rarities. Fibroma, melanotic sarcoma and secondary deposits from other tumours have been reported. Either papilloedema followed by optic atrophy or atrophy alone may be found. *Exophthalmos*
Other tumours

Bone tumours arise occasionally within the orbit and lead to optic nerve damage from pressure. The frontal sinus is a relatively common site from which secondary invasion of the orbit may arise. Angiomas, which may be either cavernous or telangiectatic, are seen from time to time, but are not common.

The precise diagnosis of tumours of the optic nerve or of new growths within the orbit is fraught with difficulty. The cases have usually to be kept under very careful observation before the diagnosis can be established and active treatment undertaken. The history and age group and the elimination of thyrotoxicosis affecting only one eye assist in establishing the general *Differential diagnosis*
Monocular thyrotoxicosis

diagnosis. The precise nature of the new growth may be established only by operation and subsequent histological study.

7. OPERATIONS

Removal of intra-orbital tumours may be carried out by two methods.

(1) Krönlein's operation

The classical operation is that of Krönlein, in which a portion of the lateral wall of the orbit is removed in order to expose the contents. In suitably selected cases, this procedure is still appropriate and is less perilous than the intracranial approach.

(2) Naffziger's operation

The second operation is associated with the name of Naffziger, and falls more within the province of the neurological surgeon than that of the ophthalmic surgeon. It was introduced originally for the relief of patients suffering from malignant exophthalmos, and consists essentially of removing the roof of the orbit from the intracranial aspect, producing a transfrontal decompression. This gives a better exposure of the intra-orbital contents, and has been used more frequently in recent years than operations of the Krönlein type. It can be carried out with safety only with the specialized technique and equipment of the neurological surgeon.

*Intracranial
approach*

Alternative method.—The alternative to operations of these two types involves removal of the eye and in many cases the major portion of the orbital contents. Since the conditions described are all of slow growth, relatively low malignancy and only rarely involve the globe, it is highly desirable to make every effort to save the eyeball, for cosmetic reasons.

BIBLIOGRAPHY AND REFERENCES

- Ballantyne, A. J. (1943). *Brit. J. Ophthalmol.*, 27, 383.
 Berens, C. (1936). *The Eye and its Diseases*. London; Saunders.
 Stallard, H. B. (1946). *Eye Surgery*. Bristol; Wright.
 Wolff, E. (1940). *The Anatomy of the Eye and Orbit, including the Central Connections, Development, and Comparative Anatomy of the Visual Apparatus*, 2nd ed. London; Lewis.

 *Treatment, Forensic*
 Clampton. St. Louis;
 Mosby.

[References to other titles are given under Optic Nerve in the Index Volume.]

ORBIT—INJURIES, INFECTIONS, NEOPLASMS

By E. F. KING, M.B., F.R.C.S., D.O.M.S.

OPHTHALMIC SURGEON, WESTMINSTER HOSPITAL; SURGEON, MOORFIELDS,
WESTMINSTER AND CENTRAL EYE HOSPITAL, LONDON

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1. DEFINITION

250.] The orbits are two bony cavities, each approximately the shape of a pear, on either side of the root of the nose. Within the cavity lie the globe

and orbital part of the optic nerve, extra-ocular muscles, lacrimal gland, arteries, veins, nerves and a mass of fibro-fatty tissue.

2. SURGICAL ANATOMY

(1) Bony orbit

Medial wall

The bony orbit consists of a roof, a floor, and medial and lateral walls, each composed of thin bone which provides only a slender barrier from surrounding structures. The medial wall lies in the sagittal plane, and the lateral wall is directed forwards and outwards from it at an angle of 45 degrees. It follows that in the operation of excision of the globe a greater length of the optic nerve can be secured by inserting the scissors in an antero-posterior direction along the inner wall of the orbit.

Margin

The margin of the orbit somewhat overhangs the cavity, thereby offering protection to its contents. To the outer side, however, it curves backwards, in order to increase the lateral field of vision, and it is from this direction that the globe is most vulnerable to direct trauma.

Roof

In the roof are seen antero-laterally the fossa for the lacrimal gland and antero-medially a depression for the trochlea, or pulley, through which passes the superior oblique muscle. The latter is readily injured, with resulting troublesome diplopia, in exploration of the orbit and operations on the frontal sinus. The floor is traversed by the infra-orbital sulcus and canal, which carry the infra-orbital nerve. From the floor of this canal the alveolar canals offer a potential path for spread of infection from the teeth to the orbit.

Orbital fissures

Above and below the lateral wall, between it and the roof and floor respectively, in the posterior part of the orbit, are the superior and inferior orbital fissures. The former opens into the middle cranial fossa and transmits the main nerves and vessels to the orbit and globe; the latter communicates with

(2) Periorbita

Where the optic canal enters the orbit the dura mater divides to form the sheath of the orbital portion of the optic nerve and the periorbita, or periosteum lining the orbit. This is loosely attached and is readily stripped up by effusions or at operation.

(3) Veins

There are three main venous outlets from the orbit.

(i) Forwards, through anastomoses with the angular veins, to the facial system.

(ii) Downwards to the pterygoid plexus and so to the jugular vein.

(iii) Backwards to the cavernous sinus.

There is thus free communication with the superficial and deep veins of the face and nasal cavities and with the venous sinuses within the skull.

(4) Lymphatics

It is noteworthy that no lymphatic glands or vessels are found in the orbit.

(5) Relations

The important anatomical relations to the orbit are as follows.

- (i) *Above*: the frontal sinus and anterior cranial fossa of the skull.
- (ii) *Medially*: the ethmoid cells anteriorly and the sphenoidal sinus posteriorly.
- (iii) *Below*: the maxillary antrum.
- (iv) *Laterally*: the middle cranial fossa of the skull and the temporal and pterygo-palatine fossae.

The intimate relation of the paranasal sinuses to three of the orbital walls is of particular significance.

3. INJURIES INVOLVING THE ORBIT

(1) Perforating injuries

Sharp instruments, twigs or other foreign bodies may penetrate the lids or conjunctival fornices and enter the orbit, causing haemorrhage or localized infection. The tip of the penetrating agent may break off and its presence in the orbit be overlooked, particularly if the path of entry is not obvious.

(2) Concussion injuries

A retro-ocular haemorrhage, which usually absorbs readily and completely, may result from this type of injury. Proptosis can be severe and prevent complete closure of the lids, which in turn leads to drying of the cornea and to exposure keratitis. Such a complication demands exploration of the orbit, evacuation of some of the blood and tarsorrhaphy of the lid margins.

(3) Fractures of the bony orbit

(See Facio-maxillary Injuries, Vol. 4, p. 15.)

The roof of the orbit may be involved in a fracture of the anterior cranial fossa, but the great majority of fractures of the orbit are the result of direct violence to the upper part of the face.

These fractures may be considered under the following headings.

(a) *Superficial*

The bony margins of the orbit, and sometimes also the nasal bones and zygomatic arch, are fractured, but the orbital walls escape. Displacement of bony fragments is slight and treatment presents little difficulty.

(b) *Deep*

The walls of the orbit are involved and there are usually associated fractures of the face and the paranasal sinuses. Displacement of bony fragments is marked, and the ultimate deformity may be considerable.

Three main types of such fracture are recognized:

- (i) *A central fracture*, involving the medial wall of both orbits, the nasal arch and the maxilla.
- (ii) *A lateral fracture*, involving the lateral wall and floor of one orbit and the neighbouring malar bone.
- (iii) *A depressed fracture* of the orbital floor, without implication of the orbital margin or facial bones.

The treatment of these fractures lies more properly within the scope of the plastic surgeon. Early reduction of bony deformities is essential, and fixation

may be attained only by indirect attachment to dental splints or to a plaster-of-Paris skull-cap.

(4) Residual diplopia

The ophthalmic surgeon may be asked to deal with a residual diplopia, due to direct damage to one or more of the extra-ocular muscles or to its nerve supply. This troublesome condition may be treated by: (i) Operative adjustment of one or more of the extra-ocular muscles, in either or both eyes, to restore parallelism of the visual axes. (ii) If the globe has dropped, a false floor to the orbit—either a graft from the iliac crest or a plate of plastic material—can be inserted beneath the orbital fat. (iii) Orthoptic exercises may help the patient to overcome a minor deviation of the visual axes.

(5) Clinical investigation

(a) Symptoms and signs

The following symptoms and signs, some of which may be elicited immediately and others only after an interval, should be considered after any injury involving the orbit.

(i) *Damage to the eye and optic nerve* is of first importance in regard to diagnosis and treatment, but is not under review in this article.

(ii) *Ecchymosis* of the lids and subconjunctival haemorrhage, particularly when first appearing some hours after the injury, suggest an underlying fracture. Swelling of the lids may render examination difficult.

(iii) *Surgical emphysema* of the lids, more marked after blowing the nose, indicates a fracture of the orbital walls and communication with the paranasal sinuses, usually the ethmoid cells. The possibility of confusion with orbital cellulitis due to gas-producing organisms must be kept in mind.

(iv) *Loss of continuity* of the orbital margin, local tenderness and displacement of bony fragments are indicative of fracture.

(v) *Anaesthesia* of the skin of the lids may result from damage to branches of the trigeminal nerve, particularly the infra-orbital branch. Local contusion may render this sign inconclusive.

(vi) *Diplopia* may be early and transient (due to orbital haemorrhage or bruising of the extra-ocular muscles), or permanent (the result of displacement of the globe following fracture of the orbital walls).

(vii) *Proptosis* of immediate onset is the result of retro-ocular haemorrhage or displacement of the globe by bony fragments. When first seen some weeks or months after the injury, an arteriovenous aneurysm, occasionally of the orbital vessels though more commonly between the internal carotid artery and cavernous sinus, is the explanation. In such a case pulsation of the globe with a bruit can be elicited and the veins of the lids and retina are congested.

(viii) *Enophthalmos*, sometimes with associated downward displacement of the globe, indicates a fracture with depression of the orbital floor.

(ix) *Cellulitis from a foreign body*. A retained orbital foreign body is suggested by intractable orbital cellulitis, particularly if a sinus is present. Probing and x-ray examination may assist the diagnosis.

(x) *Traumatic stricture of the naso-lacrimal duct*, with secondary infection of the lacrimal sac, sometimes complicates fractures of the maxilla and inner orbital wall.

(b) Radiography

X-ray examination is indicated in all cases. The radiographic technique required to display fractures of the orbital walls, and to define the displacement of the bony fragments, is highly specialized.

4. INFLAMMATORY DISEASES OF THE ORBIT

(1) Periostitis

The periorbital, or periosteum of the orbit, is continuous anteriorly with that of the facial bones and posteriorly with the dural sheath of the optic nerve.

(a) Causes

These include: (i) *injury*, especially of the orbital margin; (ii) *direct spread* from the paranasal sinuses; (iii) *primary disease* of the underlying bone, particularly tuberculous infection in children and syphilitic infection in adults.

(b) Signs and symptoms

(i) *Superficial periostitis*.—Pain is often severe, particularly at night, and is associated with local oedema of the lid, a tender swelling of the overlying skin and abscess formation; the abscess may burst superficially. The end-result may be a persistent sinus, in which bare bone is felt and from which necrosed bone may be extruded, or a depressed cicatrix, in which the skin is attached to the underlying periosteum. X-ray examination may reveal a localized area of bone rarefaction.

(ii) *Deep periostitis*.—The clinical picture tends to be less well defined than the more superficial infections. Pain is essentially deep-seated and may be referred to the supra-orbital and temporal regions. Constitutional disturbance is often severe. If local swelling is marked there may be displacement of the globe or limitation of its movements. Pus in the roof of the orbit may discharge into the anterior cranial fossa, giving rise to meningitis or cerebral abscess.

(c) Treatment

This must be considered from three points of view.

(i) *Treatment of the primary cause*.—In the majority of cases this is infection of the paranasal sinuses, which must be dealt with alone or in conjunction with exploration of the orbit.

(ii) *General treatment*.—In particular, systemic penicillin and the sulphonamides in full dosage are indicated.

(iii) *Surgical intervention*.—Most cases subside with general treatment and attention to the primary cause. The following are indications for operation, *Indications for operation* though whenever possible time should be allowed for the establishment of local tissue resistance: (1) severe and increasing constitutional disturbance, particularly when the roof of the orbit is involved, owing to the risk of secondary intracranial infection; (2) obvious and uncontrolled local spread of the infection; (3) superficial abscess formation, which will require incision and drainage; (4) a persistent sinus, for the removal of necrosed bone.

(2) Orbital cellulitis

Inflammation of the fibro-adipose tissue of the orbit is usually acute, ending in suppuration, though subacute and chronic cases are seen. It is commonly confined to one orbit.

(a) Causes

(i) *Trauma*.—Cellulitis may occur as a result of penetrating wounds, particularly if a foreign body is retained within the orbit. Infection may on occasions spread to the orbit, by the way of the alveolar canals, from an infected socket following extraction of a tooth.

(ii) *Direct spread*.—By far the commonest cause of orbital cellulitis is extension from infection in the paranasal sinuses, the bony walls of separation being thin and perforated by many foramina. The sinuses most commonly responsible are, in order of frequency, the ethmoids, frontal sinus, antrum, and sphenoidal air sinus. Infection may spread to the orbit from an osteomyelitis of the maxilla or the frontal bone, particularly in children.

It is noteworthy that the orbit is very rarely secondarily involved in infections of the face, lids or nasopharynx.

(iii) *Metastatic infection*.—Infection of the orbital tissues via the blood stream is rare but may be seen in the infective fevers or in any type of pyaemia, more particularly that occurring in post-operative abdominal infection. Occasionally a boil, or some other trivial infection, may be the primary focus.

(b) Signs and symptoms

Pain is deep-seated and is increased by movement of the globe. The constitutional reaction is severe. The lids are swollen, the conjunctiva oedematous and the globe proptosed. Infection may spread into the globe, leading to panophthalmitis, or keratitis may ensue from exposure of the cornea. Meningitis, cerebral abscess and cavernous sinus thrombosis may occur as complications.

A chronic and slowly progressive type of orbital cellulitis, known as inflammatory pseudo-tumour of the orbit, is occasionally seen.

(c) Differential diagnosis

(i) *Cavernous sinus thrombosis*.—The following points are suggestive of thrombosis of the cavernous sinus: (1) constitutional reaction of great severity; (2) extra-ocular palsies, particularly of the fourth and sixth cranial nerves, though these may be difficult to elicit owing to proptosis and impaired movements of the globe; (3) oedema over the mastoid process; (4) congestion of the retinal veins. It must be remembered, however, that orbital cellulitis and cavernous sinus thrombosis may both be present.

(ii) *Tenonitis*.—This is a subacute infection of the fascial sheath, usually of unknown aetiology, which envelops the globe, and blends anteriorly with the insertion of the ocular muscles and posteriorly with the sheath of the optic nerve. There is intense congestion and tenderness of the globe, but the constitutional disturbance, oedema of the lids, proptosis and limitation of ocular movements are minimal.

(iii) *Panophthalmitis*.—The cornea is hazy, hypopyon (pus in the anterior chamber) may be present and a yellow reflex may be seen in the pupil.

indicating infection in the vitreous. The lids show some oedema but there is no proptosis or limitation of ocular movements.

(iv) *Suppurative conditions of lids and conjunctiva.*—Oedema of the lids and discharge are often marked, but the globe is not displaced nor are its movements restricted.

(d) Treatment

The methods and indications for treatment outlined above for periostitis apply equally to orbital cellulitis. The paranasal sinuses, again, are the commonest primary source of infection and demand first attention in investigation and treatment. Surgical exploration of the orbit, to localize and drain pus, is more frequently indicated than with periostitis, but the prognosis is improved if sufficient time has elapsed to establish local resistance in the region of the focus of infection.

5. TUMOURS OF THE ORBIT

Tumours in the orbit are rare. They may be considered as benign or malignant

(1) Benign tumours

(i) *Dermoid cysts* are congenital tumours, which often increase in size at or about puberty. They are lined by stratified epithelium in which are sebaceous glands and hair follicles, and are filled with sebaceous material. Clinically, they present as cystic swellings, usually beneath the outer or inner end of the upper lid. The differential diagnosis from meningo-encephalocele is important. In the latter the swelling is invariably attached to the bone, in which a hole may be palpable or demonstrable by radiography; pulsation is present, synchronous with the pulse and respiration; the lump may be diminished in size by firm pressure, and clear cerebrospinal fluid is obtained on exploratory puncture. Differential diagnosis

(ii) *Dermo-lipomas* are congenital tumours consisting of a mass of fibro-fatty tissue which is not encapsulated. They present as diffuse swellings between the globe and outer canthus of the lids and extend deeply into the orbit.

(iii) *Tumours of the lacrimal gland*, which resemble those found in the parotid gland, are rare. Adenocarcinoma, which may be locally malignant, and endothelioma, a mixed tumour containing cartilage and myxomatous material, are seen. The differential diagnosis from dermoid cysts must be established.

(iv) *Osteomas* usually arise in the frontal or ethmoid sinuses; by erosion of the bony walls they invade the orbit. The bone formation is intensely hard and may attain considerable size, leading to displacement of the globe.

(v) *Angiomas*. Cavernous haemangiomas are encapsulated tumours of slow growth, found near the optic nerve though not attached to it. They are relatively common and can be readily removed.

Venous haemangiomas or varicosity of the orbital veins may lead to atrophy of the orbital fat, and so to enophthalmos when the head is erect. When, however, the head is bent forwards or pressure is applied to the jugular vein, the vessels become congested and the eye proptosed.

(vi) *Neurofibromas*. A single isolated neurofibroma is occasionally found within the orbit. More commonly a diffuse neurofibromatous mass, in which enlarged and irregular nerve cords can be felt, invades the orbit, the lids and

sometimes the temporal region. The ciliary nerves within the globe and orbit may also be involved.

(vii) *Hydatid cysts* are not strictly neoplastic, though it may be noted here that hydatid and other parasitic cysts may be found in the orbit.

(2) Malignant tumours

(i) *Sarcomas*, which may arise from the periosteum or the soft tissues of the orbit, are usually seen in children and are of intense malignancy.

(ii) *Carcinomas* may be the result of direct spread from the paranasal sinuses, nasopharynx or lacrimal gland, or may be a metastatic deposit, usually from a primary growth in the breast.

(iii) *Malignant melanoma of the choroid* and *retinoblastoma* sometimes extend outside the globe and invade the orbit.

(iv) *Lymphomas*, *lymphosarcomas*, *chibromas* and *orbital deposits* in association with *leukaemia* are rare; they tend to affect both orbits.

(v) *Tumours of the optic nerve*, though not primarily orbital, manifest themselves clinically as such. They are commonly *endotheliomas* arising from the dura, or from septa within the nerve derived from the pia arachnoid; tumours of the nerve tissues are exceptional. Pressure on the nerve occurs, with impairment of central or peripheral vision, and though these tumours are not of high malignancy intracranial extension by way of the optic canal may ensue.

(3) Signs and symptoms

(i) *Proptosis*, which increases in relation to the growth of the neoplasm, is the constant clinical finding in benign and malignant tumours. If the mass is within the cone of muscles—that is to say, is embraced by the extra-ocular muscles—the proptosis is mainly forwards; if it is outside the cone of muscles the globe will be displaced away from the side of the tumour, as well as forwards.

(ii) *Limitation of movement* of the globe in all directions occurs if the proptosis is severe; if the globe is displaced to one side there will be impairment of mobility in the opposite direction. Movements of the globe may also be affected by paralysis of one or more of the extra-ocular muscles, due to pressure by the tumour on their motor nerves.

(iii) *Pain*, which may be referred in distribution, is a feature of malignant growths and is the result of pressure on branches of the trigeminal nerve.

(iv) *Oedema of the lids* indicates a rapidly growing tumour, usually malignant.

(v) *Exposure keratitis*, which may lead to perforation of the globe, occurs if the proptosis is sufficient to prevent effective closure of the lids over the cornea.

(vi) *A swelling* may be palpable, particularly if the tumour is in the anterior part of the orbit and below or to the outside of the globe.

(vii) *Impairment of vision* may result from pressure on the optic nerve. If the central retinal vein is compressed, retinal haemorrhages or papilloedema are seen ophthalmoscopically.

(4) Differential diagnosis

(For exophthalmos and enophthalmos, see Eye—in Relation to Endocrine Disturbance, Vol. 3, p. 489.) The diagnosis of an orbital tumour may be

obvious, though it more often presents a complex problem. The opinions of the physician, particularly to exclude thyrotoxic exophthalmos, of the rhinologist, to eliminate disease in the paranasal sinuses and nasopharynx, of the radiologist, to demonstrate shadows or rarefaction in the orbit and enlargement of the optic canal, and of the haematologist, are essential. Not infrequently a progressive proptosis is the only positive finding and the diagnosis is established by exploration.

(5) Treatment

(a) *Benign tumours*

A benign tumour within the orbit usually demands exploration, for gradual increase in size is the rule. This leads ultimately to disfigurement, pain and danger to vision from exposure of the cornea and pressure on the optic nerve.

(b) *Malignant tumours*

A malignant growth restricted to the orbit necessitates exenteration of all the orbital contents, followed by radiotherapy.

A secondary carcinomatous deposit indicates general dissemination of the disease, and local removal is not required, particularly as these metastases can be controlled for a time by radiotherapy. An irradiation cataract will probably develop after some months, but this is of small practical importance in a patient whose expectation of life is severely limited.

Malignant growths extending into the orbit from the sinuses or nasopharynx require the co-operation of the ophthalmic surgeon and the rhinologist. Radical removal of the primary growth and orbital contents is occasionally possible, though more often treatment is confined to palliative radiotherapy.

6. OPERATIONS

(1) Orbitotomy

The orbit may be explored by the anterior, lateral or transfrontal approach.

(a) *Anterior orbitotomy*

Access to the orbit is limited in this approach, which will suffice only for exploration of inflammatory foci or the removal of neoplasms situated anteriorly.

The site of operation will be dictated by that of the lesion, but whenever possible the incision should follow the bony margin of the orbit through the medial or lateral thirds of the lids. The middle third of the upper lid must be avoided in view of the risk to the levator palpebrae superioris, with resulting ptosis. The site of the trochlea of the superior oblique muscle, at the junction of the roof and medial walls of the orbit anteriorly, must be remembered.

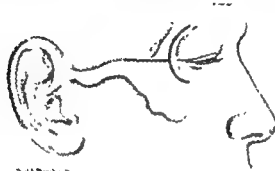
The skin, orbicularis muscle and septum orbitale are divided, and the orbital fat is reached. After the necessary manoeuvres the orbit is drained, if required, and the wound closed by the usual deep and superficial sutures.

(b) *Lateral orbitotomy*

In this operation, associated with Krönlein's name, a large part of the malar bone and lateral orbital wall are mobilized and lifted aside to give access to the orbit. Generous exposure of the whole cavity, the globe and the orbital part of the optic nerve is secured. The ultimate cosmetic result is good.

Incision

The skin incision is in two parts, which follow the outer margin of the orbit and the upper border of the zygomatic arch (Fig. 204). The temporal fascia is divided from the posterior lip of the orbital margin and the muscle stripped from the anterior part of the temporal fossa. The periosteum is next reflected from the superficial and deep surfaces of the malar bone. The periorbita is now incised along the anterior edge of the orbital margin and



Judd/Reichardt

FIG. 204—Skin incisions in Krönlein's lateral orbitotomy.



FIG. 205—Division of bones in Krönlein's lateral orbitotomy.

together with the orbital contents, is displaced medially until the inferior orbital fissure is identified.

Division of bone

A Gigli saw, an osteotome or Albee's electric saw may be used to divide the bone in three places: (i) from the zygomatic process of the frontal bone to the middle of the inferior orbital fissure, (ii) from the lower and outer angle of the orbital margin backwards and inwards to the anterior end of the inferior orbital fissure, and (iii) across the zygomatico-temporal suture (Fig. 205). The irregular mass of bone thus separated can be hinged backwards or completely removed during the exploration of the orbit.

Exploration

The periorbita is now incised and the lateral rectus identified and retracted. Exploration of the orbit should be carried out with the forefinger and by blunt dissection.

Closure

The operation is completed by closure of the periorbita and replacement of the bony fragment, which is secured by deep stitches through the soft structures. Drainage is not usually required. The wound is closed in two layers.

(c) Transfrontal orbitotomy

This approach is of value when exploration of the anterior cranial fossa and optic canal, as well as of the orbit, is required.

The operation, in which the skull is opened through the frontal bone, the frontal lobes are lifted and the orbital roof is removed, is essentially in the province of the neuro-surgeon.

(2) Exenteration of the orbit

Complete removal of the orbital contents is required for malignant disease, and to offer facilities for irradiation of neoplasms in the nasal sinuses.

The outer canthus of the lids is divided down to the bony margin. The conjunctiva is next incised throughout the length of the upper and lower fornices, the two incisions joining at the inner and outer sides of the globe. The whole of the contents are now separated from the orbital walls by blunt dissection as far as the apex of the orbit, where they are divided by scissors. Severe haemorrhage, which can be controlled only by diathermy coagulation of the soft tissues, may be met at this stage. The lash-bearing margin and the conjunctiva lining the posterior surface are removed from both lids. *Technique*

Grafting of the socket is not necessary, for epithelization readily occurs from the skin of the lids which remains.

BIBLIOGRAPHY

1. J. H. J. (1900) *The Eye*. London: Baillière Tindall.
 2. J. H. J. (1901) *The Eye*. London: Baillière Tindall.
 3. J. H. J. (1902) *The Eye*. London: Baillière Tindall.
 4. J. H. J. (1903) *The Eye*. London: Baillière Tindall.
 5. J. H. J. (1904) *The Eye*. London: Baillière Tindall.
 6. J. H. J. (1905) *The Eye*. London: Baillière Tindall.
 7. J. H. J. (1906) *The Eye*. London: Baillière Tindall.
 8. J. H. J. (1907) *The Eye*. London: Baillière Tindall.
 9. J. H. J. (1908) *The Eye*. London: Baillière Tindall.
 10. J. H. J. (1909) *The Eye*. London: Baillière Tindall.
 11. J. H. J. (1910) *The Eye*. London: Baillière Tindall.
 12. J. H. J. (1911) *The Eye*. London: Baillière Tindall.
 13. J. H. J. (1912) *The Eye*. London: Baillière Tindall.
 14. J. H. J. (1913) *The Eye*. London: Baillière Tindall.
 15. J. H. J. (1914) *The Eye*. London: Baillière Tindall.
 16. J. H. J. (1915) *The Eye*. London: Baillière Tindall.
 17. J. H. J. (1916) *The Eye*. London: Baillière Tindall.
 18. J. H. J. (1917) *The Eye*. London: Baillière Tindall.
 19. J. H. J. (1918) *The Eye*. London: Baillière Tindall.
 20. J. H. J. (1919) *The Eye*. London: Baillière Tindall.
 21. J. H. J. (1920) *The Eye*. London: Baillière Tindall.
 22. J. H. J. (1921) *The Eye*. London: Baillière Tindall.
 23. J. H. J. (1922) *The Eye*. London: Baillière Tindall.
 24. J. H. J. (1923) *The Eye*. London: Baillière Tindall.
 25. J. H. J. (1924) *The Eye*. London: Baillière Tindall.
 26. J. H. J. (1925) *The Eye*. London: Baillière Tindall.
 27. J. H. J. (1926) *The Eye*. London: Baillière Tindall.
 28. J. H. J. (1927) *The Eye*. London: Baillière Tindall.
 29. J. H. J. (1928) *The Eye*. London: Baillière Tindall.
 30. J. H. J. (1929) *The Eye*. London: Baillière Tindall.
 31. J. H. J. (1930) *The Eye*. London: Baillière Tindall.
 32. J. H. J. (1931) *The Eye*. London: Baillière Tindall.
 33. J. H. J. (1932) *The Eye*. London: Baillière Tindall.
 34. J. H. J. (1933) *The Eye*. London: Baillière Tindall.
 35. J. H. J. (1934) *The Eye*. London: Baillière Tindall.
 36. J. H. J. (1935) *The Eye*. London: Baillière Tindall.
 37. J. H. J. (1936) *The Eye*. London: Baillière Tindall.
 38. J. H. J. (1937) *The Eye*. London: Baillière Tindall.
 39. J. H. J. (1938) *The Eye*. London: Baillière Tindall.
 40. J. H. J. (1939) *The Eye*. London: Baillière Tindall.
 41. J. H. J. (1940) *The Eye*. London: Baillière Tindall.
 42. J. H. J. (1941) *The Eye*. London: Baillière Tindall.
 43. J. H. J. (1942) *The Eye*. London: Baillière Tindall.
 44. J. H. J. (1943) *The Eye*. London: Baillière Tindall.
 45. J. H. J. (1944) *The Eye*. London: Baillière Tindall.
 46. J. H. J. (1945) *The Eye*. London: Baillière Tindall.
 47. J. H. J. (1946) *The Eye*. London: Baillière Tindall.
 48. J. H. J. (1947) *The Eye*. London: Baillière Tindall.
 49. J. H. J. (1948) *The Eye*. London: Baillière Tindall.
 50. J. H. J. (1949) *The Eye*. London: Baillière Tindall.
 51. J. H. J. (1950) *The Eye*. London: Baillière Tindall.
 52. J. H. J. (1951) *The Eye*. London: Baillière Tindall.
 53. J. H. J. (1952) *The Eye*. London: Baillière Tindall.
 54. J. H. J. (1953) *The Eye*. London: Baillière Tindall.
 55. J. H. J. (1954) *The Eye*. London: Baillière Tindall.
 56. J. H. J. (1955) *The Eye*. London: Baillière Tindall.
 57. J. H. J. (1956) *The Eye*. London: Baillière Tindall.
 58. J. H. J. (1957) *The Eye*. London: Baillière Tindall.
 59. J. H. J. (1958) *The Eye*. London: Baillière Tindall.
 60. J. H. J. (1959) *The Eye*. London: Baillière Tindall.
 61. J. H. J. (1960) *The Eye*. London: Baillière Tindall.
 62. J. H. J. (1961) *The Eye*. London: Baillière Tindall.
 63. J. H. J. (1962) *The Eye*. London: Baillière Tindall.
 64. J. H. J. (1963) *The Eye*. London: Baillière Tindall.
 65. J. H. J. (1964) *The Eye*. London: Baillière Tindall.
 66. J. H. J. (1965) *The Eye*. London: Baillière Tindall.
 67. J. H. J. (1966) *The Eye*. London: Baillière Tindall.
 68. J. H. J. (1967) *The Eye*. London: Baillière Tindall.
 69. J. H. J. (1968) *The Eye*. London: Baillière Tindall.
 70. J. H. J. (1969) *The Eye*. London: Baillière Tindall.
 71. J. H. J. (1970) *The Eye*. London: Baillière Tindall.
 72. J. H. J. (1971) *The Eye*. London: Baillière Tindall.
 73. J. H. J. (1972) *The Eye*. London: Baillière Tindall.
 74. J. H. J. (1973) *The Eye*. London: Baillière Tindall.
 75. J. H. J. (1974) *The Eye*. London: Baillière Tindall.
 76. J. H. J. (1975) *The Eye*. London: Baillière Tindall.
 77. J. H. J. (1976) *The Eye*. London: Baillière Tindall.
 78. J. H. J. (1977) *The Eye*. London: Baillière Tindall.
 79. J. H. J. (1978) *The Eye*. London: Baillière Tindall.
 80. J. H. J. (1979) *The Eye*. London: Baillière Tindall.
 81. J. H. J. (1980) *The Eye*. London: Baillière Tindall.
 82. J. H. J. (1981) *The Eye*. London: Baillière Tindall.
 83. J. H. J. (1982) *The Eye*. London: Baillière Tindall.
 84. J. H. J. (1983) *The Eye*. London: Baillière Tindall.
 85. J. H. J. (1984) *The Eye*. London: Baillière Tindall.
 86. J. H. J. (1985) *The Eye*. London: Baillière Tindall.
 87. J. H. J. (1986) *The Eye*. London: Baillière Tindall.
 88. J. H. J. (1987) *The Eye*. London: Baillière Tindall.
 89. J. H. J. (1988) *The Eye*. London: Baillière Tindall.
 90. J. H. J. (1989) *The Eye*. London: Baillière Tindall.
 91. J. H. J. (1990) *The Eye*. London: Baillière Tindall.
 92. J. H. J. (1991) *The Eye*. London: Baillière Tindall.
 93. J. H. J. (1992) *The Eye*. London: Baillière Tindall.
 94. J. H. J. (1993) *The Eye*. London: Baillière Tindall.
 95. J. H. J. (1994) *The Eye*. London: Baillière Tindall.
 96. J. H. J. (1995) *The Eye*. London: Baillière Tindall.
 97. J. H. J. (1996) *The Eye*. London: Baillière Tindall.
 98. J. H. J. (1997) *The Eye*. London: Baillière Tindall.
 99. J. H. J. (1998) *The Eye*. London: Baillière Tindall.
 100. J. H. J. (1999) *The Eye*. London: Baillière Tindall.
 101. J. H. J. (2000) *The Eye*. London: Baillière Tindall.
 102. J. H. J. (2001) *The Eye*. London: Baillière Tindall.
 103. J. H. J. (2002) *The Eye*. London: Baillière Tindall.
 104. J. H. J. (2003) *The Eye*. London: Baillière Tindall.
 105. J. H. J. (2004) *The Eye*. London: Baillière Tindall.
 106. J. H. J. (2005) *The Eye*. London: Baillière Tindall.
 107. J. H. J. (2006) *The Eye*. London: Baillière Tindall.
 108. J. H. J. (2007) *The Eye*. London: Baillière Tindall.
 109. J. H. J. (2008) *The Eye*. London: Baillière Tindall.
 110. J. H. J. (2009) *The Eye*. London: Baillière Tindall.
 111. J. H. J. (2010) *The Eye*. London: Baillière Tindall.
 112. J. H. J. (2011) *The Eye*. London: Baillière Tindall.
 113. J. H. J. (2012) *The Eye*. London: Baillière Tindall.
 114. J. H. J. (2013) *The Eye*. London: Baillière Tindall.
 115. J. H. J. (2014) *The Eye*. London: Baillière Tindall.
 116. J. H. J. (2015) *The Eye*. London: Baillière Tindall.
 117. J. H. J. (2016) *The Eye*. London: Baillière Tindall.
 118. J. H. J. (2017) *The Eye*. London: Baillière Tindall.
 119. J. H. J. (2018) *The Eye*. London: Baillière Tindall.
 120. J. H. J. (2019) *The Eye*. London: Baillière Tindall.
 121. J. H. J. (2020) *The Eye*. London: Baillière Tindall.
 122. J. H. J. (2021) *The Eye*. London: Baillière Tindall.
 123. J. H. J. (2022) *The Eye*. London: Baillière Tindall.
 124. J. H. J. (2023) *The Eye*. London: Baillière Tindall.
 125. J. H. J. (2024) *The Eye*. London: Baillière Tindall.
 126. J. H. J. (2025) *The Eye*. London: Baillière Tindall.

[References to other titles are given under Orbit—Injuries, Infections, Neoplasms, in the Index Volume.]

ORTHODONTICS

By HAROLD CHAPMAN, F.D.S.

DENTAL SURGEON, LONDON HOSPITAL; LECTURER IN ORTHODONTICS,
LONDON HOSPITAL DENTAL SCHOOL

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1. DEFINITION

251.] Orthodontics is the branch of dentistry concerned with irregularities of the teeth. It has been described by Angle (1900) as the science of occlusion. Bennett (1931) defines occlusion as "the relationship of the teeth of the maxillae and mandible when the jaws are closed and the condyles are at rest in the glenoid fossae". The term, normal occlusion, is used to include the size, shape and relationship of the dental arches, also the alignment of the teeth and their relation to one another in the same and in opposite arches. It follows that normal occlusion refers to "straight teeth" and mal-occlusion to "irregular teeth", the latter including mal-relation to one another of the dental arches and of the jaws. In this context the terms mal-occlusion and mal-relation are used synonymously.

2. NORMAL AND ABNORMAL OCCLUSION

(1) Normal occlusion

This has been defined as an "infinite array of variants". This is particularly so regarding normal occlusion in children from 2 to 14 years of age when these variants are especially in evidence, and even more so between the ages of 6 and 12 years when the dentition consists of both deciduous and permanent teeth. In order to make a diagnosis in patients under 14 years of age, a knowledge of these variants and of certain changes is essential. Many cases were considered abnormal because, at the particular stage of development, they were not known to be variants of the normal. Had such conditions persisted until 12 years of age they would have been definitely abnormal; for example, mal-alignment of the lower incisors at 7 or 8 years of age may be a variant of normal but at 12 years the same condition is mal-occlusion. Not all of the variants are observed in one individual. The normal in the adult, from 14 years onwards, presents little difficulty; the shape of arches and other details vary, but the relationships of the teeth are constant and their alignment is satisfactory.

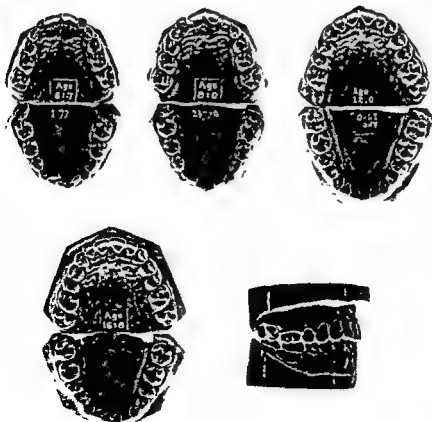
It used to be thought that the deciduous teeth were in approximal contact on eruption, and also that teeth were "straight" at all ages. The arches of deciduous teeth consist of teeth and spaces, and no deciduous tooth is in contact with another when it erupts (Fig. 206). The molar spaces close between the

FIG. 206.—(Case A 40-31; female.) Normal occlusion at age 3 years, 10 months showing spacing of all teeth; at this stage each molar occludes with only one tooth and not with two as at a later period. Photographs of models in the Odontological Section of the Museum of the Royal College of Surgeons. (By kind permission of the Hon. Curator.)

FIG. 207.—(Case 2377; female.) Normal occlusion.

The same case at four ages, showing normal

space for them; other factors may also provide some of the necessary space. The upper centrals



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*Science of
occlusion*

*Deciduous
teeth*

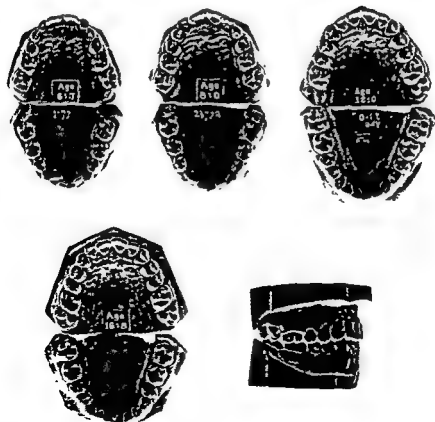
FIG. 206.—(Case A40-31: female) Normal occlusion
 Photographs of models in the Odontological

Section of the Museum of the Royal College of Surgeons. (By kind permission of the Hon. Curator.)



FIG. 207.—(Case 2377: female) Normal occlusion.
 The same case at four ages, showing normal

space for them; other factors may also provide some of the necessary space. The upper centrals are spaced; this is normal at this stage. The space may be considerably greater (as much as 3 mm.). It has usually closed by the age of 10 years, but may do so earlier or later. There is a small proportion of cases in which the space is permanent, when it may be due to the condition of the bone between the upper central incisors.



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By HAROLD CHAPMAN, F.D.S.

DENTAL SURGEON, LONDON HOSPITAL; LECTURER IN ORTHODONTICS,
LONDON HOSPITAL DENTAL SCHOOL

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(1) Normal occlusion

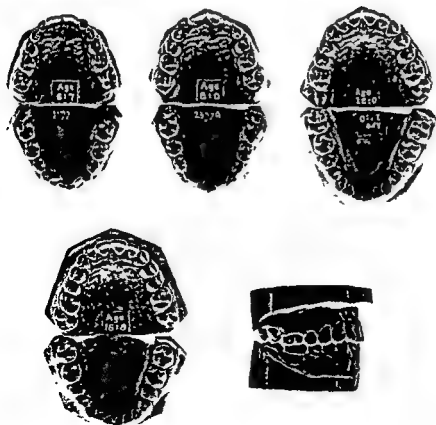
This has been defined as an "infinite array of variants". This is particularly so regarding normal occlusion in children from 2 to 14 years of age when these variants are especially in evidence, and even more so between the ages of 6 and 12 years when the dentition consists of both deciduous and permanent teeth. In order to make a diagnosis in patients under 14 years of age, a knowledge of these variants and of certain changes is essential. Many cases were considered abnormal because, at the particular stage of development, they were not known to be variants of the normal. Had such conditions persisted until 12 years of age they would have been definitely abnormal; for example, mal-alignment of the lower incisors at 7 or 8 years of age may be a variant of normal but at 12 years the same condition is mal-occlusion. Not all of the variants are observed in one individual. The normal in the adult, from 14 years onwards, presents little difficulty; the shape of arches and other details vary, but the relationships of the teeth are constant and their alignment is satisfactory.

It used to be thought that the deciduous teeth were in approximal contact on eruption, and also that teeth were "straight" at all ages. The arches of deciduous teeth consist of teeth and spaces, and no deciduous tooth is in contact with another when it erupts (Fig. 206). The molar spaces close between the

FIG 206.—(Case A40-31 : female.) Normal occlusion at age 3 years, 10 months showing spacing of all teeth; at this stage each molar occludes with only one tooth and not with two as at a later period. Photographs of models in the Odontological Section of the Museum of the Royal College of Surgeons. (*By kind permission of the Hon. Curator.*)



FIG. 207.—(Case 2377 : female.) Normal occlusion. The same case at four ages, showing normal changes (no treatment). At age 6 years, 7 months the lower permanent laterals are lingual to the line of the arch: the arch breadth increases to provide space for them. other factors may also provide some of the necessary space. The upper centrals are spaced; this is normal at this stage. The space may be considerably greater (as much as 6 mm.). It has usually closed by the age of 10 years, but may do so earlier or later. There is a small pro-



ORTHODONTICS

BY HAROLD CHAPMAN, F.D.S.

DENTAL SURGEON, LONDON HOSPITAL; LECTURER IN ORTHODONTICS,
LONDON HOSPITAL DENTAL SCHOOL

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1. DEFINITION

251.] Orthodontics is the branch of dentistry concerned with irregularities of the teeth. It has been described by Angle (1900) as the science of occlusion. Bennett (1931) defines occlusion as "the relationship of the teeth of the maxillae and mandible when the jaws are closed and the condyles are at rest in the glenoid fossae". The term, normal occlusion, is used to include the size, shape and relationship of the dental arches, also the alignment of the teeth and their relation to one another in the same and in opposite arches. It follows that normal occlusion refers to "straight teeth" and mal-occlusion to "irregular teeth", the latter including mal-relation to one another of the dental arches and of the jaws. In this context the terms mal-occlusion and mal-relation are used synonymously.

2. NORMAL AND ABNORMAL OCCLUSION

(1) Normal occlusion

This has been defined as an "infinite array of variants". This is particularly so regarding normal occlusion in children from 2 to 14 years of age when these variants are especially in evidence, and even more so between the ages of 6 and 12 years when the dentition consists of both deciduous and permanent teeth. In order to make a diagnosis in patients under 14 years of age, a knowledge of these variants and of certain changes is essential. Many cases were considered abnormal because, at the particular stage of development, they were not known to be variants of the normal. Had such conditions persisted until 12 years of age they would have been definitely abnormal; for example, mal-alignment of the lower incisors at 7 or 8 years of age may be a variant of normal but at 12 years the same condition is mal-occlusion. Not all of the variants are observed in one individual. The normal in the adult, from 14 years onwards, presents little difficulty; the shape of arches and other details vary, but the relationships of the teeth are constant and their alignment is satisfactory.

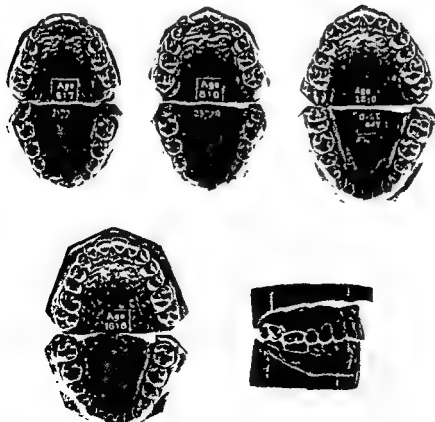
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FIG. 206.—(Case A40-31; female.) Normal occlusion at age 3 years, 10 months showing spacing of all teeth; at this stage each molar occludes with only one tooth and not with two as at a later period. Photographs of models in the Odontological Section of the Museum of the Royal College of Surgeons. (*By kind permission of the Hon Curator.*)



FIG. 207.—(Case 2377 female.) Normal occlusion. The same case at four ages, showing normal

space for them. other factors may also provide some of the necessary space. The upper centrals are spaced; this is normal at this stage. The space may be considerably greater (as much as 6 mm.) It has usually closed by the age of 10 years, but may do so earlier or later. There is a small proportion of cases in which the space is permanent, when it may be due to the condition of the bone between the upper central incisors.



ORTHODONTICS

By HAROLD CHAPMAN, F.D.S.

DENTAL SURGEON, LONDON HOSPITAL; LECTURER IN ORTHODONTICS,
LONDON HOSPITAL DENTAL SCHOOL

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I. DEFINITION

*Science of
occlusion*

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(1) Normal occlusion

This has been defined as an "infinite array of variants". This is particularly so regarding normal occlusion in children from 2 to 14 years of age when these variants are especially in evidence, and even more so between the ages of 6 and 12 years when the dentition consists of both deciduous and permanent teeth. In order to make a diagnosis in patients under 14 years of age, a knowledge of these variants and of certain changes is essential. Many cases were considered abnormal because, at the particular stage of development, they were not known to be variants of the normal. Had such conditions persisted until 12 years of age they would have been definitely abnormal; for example, mal-alignment of the lower incisors at 7 or 8 years of age may be a variant of normal but at 12 years the same condition is mal-occlusion. Not all of the variants are observed in one individual. The normal in the adult, from 14 years onwards, presents little difficulty; the shape of arches and other details vary, but the relationships of the teeth are constant and their alignment is satisfactory.

*Deciduous
teeth*

It used to be thought that the deciduous teeth were in approximal contact on eruption, and also that teeth were "straight" at all ages. The arches of deciduous teeth consist of teeth and spaces, and no deciduous tooth is in contact with another when it erupts (Fig. 206). The molar spaces close between the

important errors are those of the relation of the lower arch of teeth to the upper arch; the lower may be too far back or too far forward in relation to the upper (Figs. 209-212).

FIG. 209.—(Case 40: male)

Age 11 years, Class II, Div. I (Angle): Class III (Bennett). Good arches, indicating bones large enough to contain all the teeth in good alignment. Post-normal relation of lower to upper jaw. Compare shape of upper arch with the normal shown in Figs. 206 and 207. Figs. 209, 210, 211 and 212 are "good arches" (Figs. 209 and 210 are good as regards size but not shape), but it is common to find also "small arches" in these types, particularly in post-normal relation of the lower arch. extraction is included in the treatment in cases with small arches.

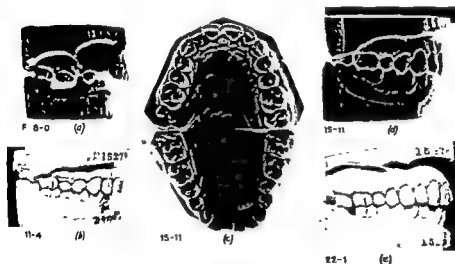
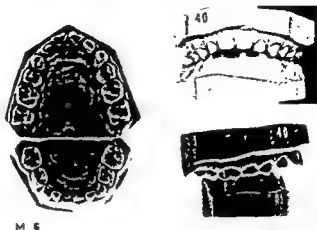


FIG. 210.—(Case 1527 female) Class II, Div. I (Angle): Class III (Bennett) Good arches, indicating bones large enough to contain all the teeth in good alignment. (a) Age 8 years before treatment (b) Age 11 years, 4 months, immediately after treatment (c) and (d) Age 15 years, 11 months, 4 years after treatment (e) The condition is similar at age 22 years, 1 month. Treatment is by means of fixed appliances to improve the alignment of the teeth, and intermaxillary traction to bring the mandible forward into correct relation with the maxillae. These cases are relatively common. The profile in these cases, and those of which Figs. 211 and 212 are examples, is improved by the changed position of the mandible, forward in one case and backward in the other.

Permanent teeth

ages of 3 and 6 years; the incisor spaces remain. The permanent lower centrals usually erupt in, or soon afterwards assume, good alignment; the permanent lower laterals may erupt lingually to the centrals and canines, the space being insufficient for them to be in alignment (Fig. 207). This gives the appearance of considerable irregularity, but subsequent arch breadth increase provides room for them and they come into line. On the other hand, the lower laterals may erupt in line with the centrals but labial to the canines (Fig. 211). The appearance is not one of such marked irregularity as the arrangement previously described, but both are the same—except for the position of the laterals in relation to the other teeth—even to the size of the space between the lower permanent centrals and the deciduous canines. The end-results will be similar though at this stage the two pictures are very dissimilar. Both arrangements of laterals may be seen in the same case, one on each side.

The upper permanent centrals always erupt spaced (Fig. 207), the interval between them being variable. In the course of time—between 2 and 8 years—the two teeth come into contact. The few exceptions to this rule are probably due to an abnormal arrangement of the bone between the teeth, and for that reason the condition may not be curable permanently. The upper arch breadth increases, the lower arch breadth also increases, but only to about half the extent of the upper. This difference of increase in arch breadth is compensated by a forward movement of the lower arch in relation to the upper one. There are numerous other variants not referred to here, a knowledge of which is essential for the diagnostician.

(2) Abnormal occlusion*Deciduous teeth*

The deciduous teeth may be in contact; this indicates that, though their alignment is perfect, the foundations which support the teeth, the maxillae and mandible are not large enough, and that in the permanent dentition in which usually there are no spaces the teeth will be crowded (Fig. 208). The other

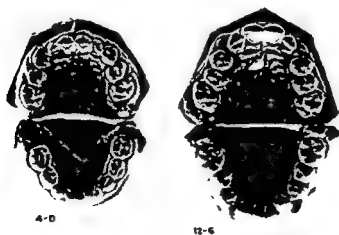


FIG. 208.—(Case 295: male.) *Class I* (Angle); *Class II* (Bennett). Maxillary bones too small for the teeth in alignment. At the age of 4 years there is a little crowding but the condition is not such as to attract attention, though, as the arches should consist of teeth and spaces, the error is actually considerable as shown at

the age 12 years, 6 months (no treatment). Good alignment of the upper teeth is obtained

the alignment of the lower incisors may be

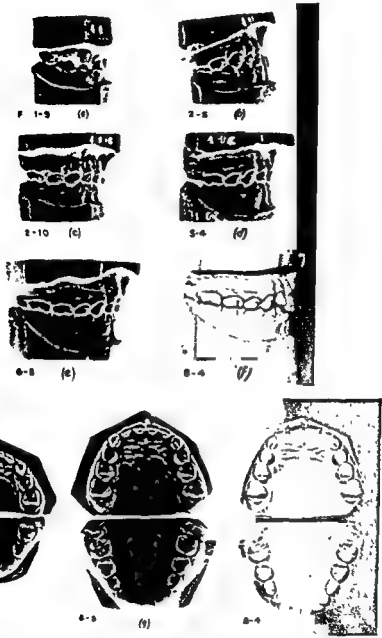


FIG. 211.—(Case L. H. 40; female) Medial relation of mandible to maxillae. Good arches. Models of original condition not available. (a) Age 1 year 9 months, after 4 months' treatment with head and chin caps to retract the mandible. Horizontal space of 3 mm. between upper and lower incisors. (b) Age 2 years 5 months, after 1 year's treatment. Upper and lower incisors in contact but their relation not corrected; the vertical incisor overlap prevented further correction, this overlap was eliminated by apparatus, correction then occurred in 10 days. (c) Age 2 years 10 months. Case corrected. Head and chin caps worn for a short time after this. Note the normal spacing of the molars. (d) Age 5 years 4 months. Molar spaces closed. Incisors spaced. (e) Age 6 years 8 months. Upper incisors spaced, all teeth deciduous except first permanent molars and lower central incisors. (f) Age 8 years 4 months. All permanent incisors erupted; the lower laterals overlap the lower canines labially (compare Fig. 207). There are no spaces. Normal occlusion. (g) Palatal aspect at ages 2 years 10 months; 6 years 8 months; and 8 years 4 months

3. AETIOLOGY OF ABNORMAL OCCLUSION

Bennett's classification (*see under* Diagnosis) indicates that causes may be grouped under the headings "local" and "general"; these may be subdivided into (1) antenatal and (2) post-natal.

*Local
causes*

Most local causes are definite. Some of the more important are as follows.

- (1) Premature loss of (Fig. 212), and prolonged retention of, deciduous teeth.
- (2) Presence of supernumerary teeth and congenital absence of the normal number of teeth.
- (3) Habit, an example of which is finger-sucking.

The effects of finger-sucking usually disappear within one year of its cessation, if this occurs not later than the age of 8-9 years.

*General
causes*

General causes are usually antenatal, the child being born with the irregularities; these include Bennett's "developmental defects of bone", which does not mean that the bone is defective as a tissue, but that the jaws are not normal as regards size, or shape or proportion, or in relation to one another (Figs. 208-212). Any combination of these defects may be present. The cause of such defects is unknown though much has been written on the subject. Evidence in support of views which have been put forward is often lacking.

Pre-normal mal-relation of the lower to the upper jaw may be brought about by habit. It is also said to occur after premature loss of all deciduous molars. In other cases this condition is believed to have an hereditary origin; an example of this is the "Hapsburg jaw".

4. DIAGNOSIS OF ABNORMAL OCCLUSION

Classification

For this purpose, cases of mal-occlusion are classified. The better-known classifications are those of Angle (1900) and of Bennett (1931). The former is based on the antero-posterior relation of the lower jaw to the upper, this being decided by the relation of the lower to the upper teeth. This relation may be (I) correct (Figs. 208 and 213), (II) post-normal or too posterior (Figs. 209 and 210), and (III) pre-normal or too anterior—that is, underhung as in a bulldog (Figs. 211 and 212). In Class II the lower front teeth are too far back, giving the appearance of prominent upper centrals; the latter may rest on the lower lip on account of its posterior position. In this class there is another type in which the upper centrals are in contact with the lower centrals, the upper laterals overlap the upper centrals, and the patient is able to keep the lips closed. Class I (Fig. 208) is a type of mal-occlusion in which the arch relation is correct; it must not, however, be confused with normal occlusion, since there may be any other irregularity present (Fig. 213).

*Dental
caries*

Bennett's classification is based on aetiology. His first class includes cases in which there is "abnormal position of one or more teeth due to local causes"; the commonest of these local causes is caries followed by premature loss of deciduous teeth, especially of the second deciduous molars. Loss of the lower molars has the more serious effect—a forward drifting of the permanent molars which reduces the space for the premolars (Fig. 212). Such effects can be prevented by care of the deciduous teeth, but if they must be lost, it may be desirable in some cases to prevent loss of space by applying a splint or space retainer which need be worn only for a part of each 24 hours. This class has no counterpart in Angle's classification; if the arch relation is correct, the case would be included in his Class I and the same may apply to Fig. 213.

*Application
of splint*

Aesthetics require good alignment of the upper front teeth and good relationship of the upper and lower incisors (Fig. 207); that is, the labial surfaces of the lower incisors should be in contact with the lingual surfaces of the upper

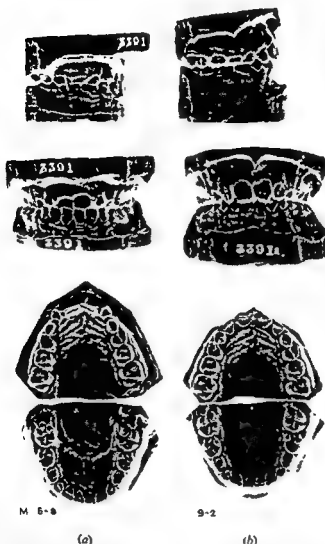


FIG. 213 — (Case 3391 male) (a) Age 6 years 8 months. Upper permanent incisors palatal or lingual to lower permanent incisors, the laterals being more so than the centrals. Upper deciduous laterals present but about to be shed. Good lower arch in correct relation with the upper arch, probably good relation of the lower incisors to the upper incisors. (b) Age 6 years 8 months. Upper permanent incisors palatal or lingual to lower permanent incisors, the laterals being more so than the centrals. Upper deciduous laterals present but about to be shed. Good lower arch in correct relation with the upper arch, probably good relation of the lower incisors to the upper incisors.

incisors. To obtain these results, the treatment of small arches will include the loss of teeth (Fig. 208). The treatment of errors of arch relation will require the relation of the mandible to the maxillae to be corrected. In a few cases of post-normal relation of the lower to the upper arch, the upper incisors may

*Develop-
mental
defects of
bones*

Bennett's Class II corresponds to Angle's Class I (Fig. 208). The feature of these cases is "abnormal formation of a part or the whole of either arch due to developmental defects of bone". Such abnormal formation is usually a smallness of the jaws in relation to the size of the teeth so that there is not room for the teeth in good alignment.

In Class III there is abnormal relationship between the upper and lower arches of the teeth. The most important relation is the antero-posterior one

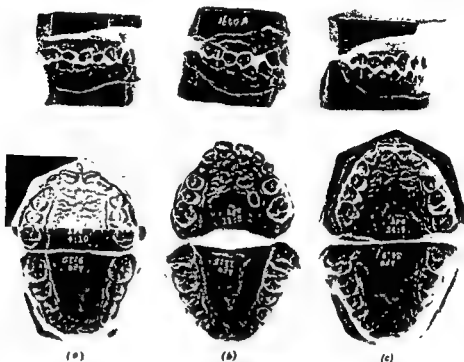


Fig. 208. (a) Normal relation of mandible to maxilla. (b) Class II relation of mandible to maxilla. (c) Class III relation of mandible to maxilla.

upper premolar has been removed to let the second premolar take its place. The first left upper permanent molar has drifted too far forward as a result of premature loss of the second left upper deciduous molar and so has reduced the space for the premolars, hence the necessity to remove one. This may occur irrespective of the size of the arches and is an entirely different reason for extraction from that referred to in Fig. 208.

and therefore this class includes Angle's Classes II and III, but its scope is wider because it includes the possibility of pre-normal or post-normal relation of the upper or lower jaws. Angle, however, considered that the upper

vertical and lateral relationship of the arches in this class is abnormal.

5. TREATMENT

The object of orthodontic treatment is to provide the child with an aesthetic and functional set of teeth. Function is rarely at a serious disadvantage.

*Aesthetic and
functional
requirements*

ORTHODONTICS—SURGERY OF

BY PATRICK CLARKSON, M.B.E., F.R.C.S.

PLASTIC SURGEON, ROYAL NORTHERN HOSPITAL, CASUALTY SURGEON,
GUY'S HOSPITAL; PLASTIC SURGEON, MINISTRY OF HEALTH PLASTIC CENTRE,
BASINGSTOKE

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1. INTRODUCTION

252.] There are a number of congenital malformations of the jaw and mal-occlusions consequent upon trauma and old infections in which the deformity is too great to respond to dental orthodontic methods alone. Surgery must be associated with orthodontic treatment, in these cases, in order to obtain the best possible function in terms of occlusion and power of mastication, as well as the closest approximation to a normal appearance. In the majority of such cases the operative procedure is localized to the facial skeleton; in others, however, an operation on bulky soft tissue or on soft-tissue scars, by relieving pressure or pull on the facial skeleton, produces the necessary result. In yet a third group the disorder in occlusion and mastication is caused by derangements of the temporo-mandibular joint, some of which can be relieved surgically. All these procedures are combined operations for surgeon and orthodontist. An essential feature is the pre-operative planning carried out with models of the jaws to determine appropriate lines of section. The post-operative care, which for a number of patients may involve several weeks in cast-metal splints with intermaxillary fixation, is principally the sphere of the orthodontist.

*Types of
operative
procedure*

In the early stages of prognathism and of recession of the jaw it is often a matter of difficulty to decide whether it is principally an overgrowth of one jaw or an undergrowth of the other which is the fault. Radiographic measurements by the cephalograph are critical for the diagnosis in some cases.

*The
cephalograph
in diagnosis*

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In the early stages of prognathism and of recession of the jaw it is often a matter of difficulty to decide whether it is principally an overgrowth of one jaw or an undergrowth of the other which is the fault. Radiographic measurements by the cephalograph are critical for the diagnosis in some cases.

*Types of
operative
procedure*

*The
cephalograph
in diagnosis*

In this chapter the methods of correction of deformities in both mandible and maxilla will be described. Details of the operative treatment of certain disorders of the temporo-mandibular joint, and of trismus, are also included, as are descriptions of some of the congenital soft-tissue deformities which can cause secondary mal-occlusions, and the operations by which these deformities are relieved. It must be emphasized again, however, that any scar about the lips and tongue, whether caused by burn or by trauma, can produce secondary effects upon the jaws. No attempt is made here at a comprehensive description of the methods of surgical relief of such scars. A final comment is made on the value of epithelial inlay in edentulous elderly patients whose alveolar ridges are too shallow to maintain their dentures in normal articulation.

2. DEFORMITIES OF THE MANDIBLE

The deformities to be described here are prognathism, gross asymmetry of the mandible, recession of the jaw, congenital open bite and mal-occlusion following mal-union of old fractures.

(1) Prognathism

Age incidence

The condition of prognathism commonly becomes apparent well before the age of 15–17 years, when growth of the jaw generally stops. By this time the

Kostecka's operation

of the lower incisors in front of the upper incisors is half a centimetre or more.

Operative technique.—Kostecka's operation is recommended for this condition. A Kostecka seeker is inserted through a stab incision half-way between the tragus of the ear and the angle of the jaw. It is passed along the deep surface of the ascending ramus in a line between the incision and the inner canthus of the eye. As the point of the seeker passes the anterior border of the



FIG. 214.—Kostecka's method of section for prognathism. Kostecka's seeker is passed deep to the mandible and then withdrawn, carrying a Gigli saw into place. The mandible is divided and displaced back into the predetermined position; it is secured by intermaxillary fixation of previously cemented cast-metal splints.

coronoid process the handle is depressed and the point is presented through a stab incision in the overlying skin of the cheek. Through the eye in the point of the seeker a stainless-steel wire is threaded and withdrawn with the seeker, carrying with it an attached Gigli saw (Fig. 214). With the saw in position the

ascending ramus of the jaw is divided, care being taken to protect the skin edges with Gillies's hooks. When both sides have been divided the horizontal ramus of the jaw is displaced backwards and secured in its new posterior position by intermaxillary fixation by wire of previously cemented cast-metal splints.

The post-operative care comprises the maintenance of intermaxillary fixation for at least 6 weeks. Subsequently flanges of the Schroeder type should be worn for 3 months before the splints are removed. *Post-operative care*

Acquired asymmetrical prognathism

This condition often follows a blow and starts later in life than does the asymmetrical prognathism described above—usually between the ages of 20

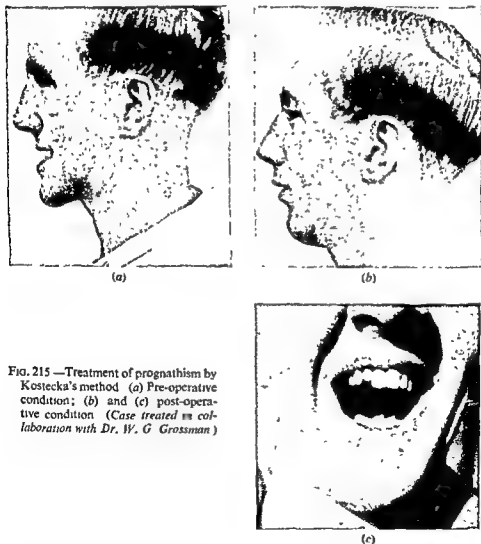


FIG. 215 —Treatment of prognathism by Kostecka's method (a) Pre-operative condition; (b) and (c) post-operative condition (Case treated in collaboration with Dr. W. G. Grossman)

and 30 years. There is an excessive overgrowth of the condyle of the jaw on one side. In addition to removal of the affected condyle, resection of the ascending ramus of the jaw on the opposite side and posterior displacement of the tooth-bearing fragment, it may be necessary to resect portions of the

tooth-bearing horizontal ramus of the jaw (Figs. 215 and 216). This can be done by the method described for the correction of congenital open bite (see p. 395).

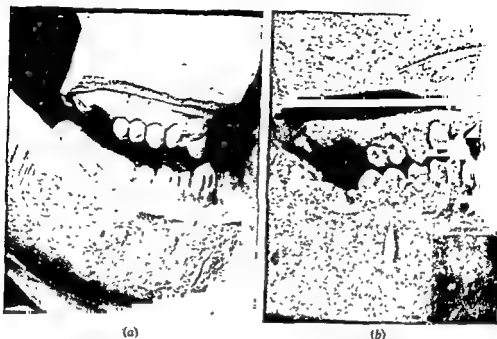


FIG. 216.—Models of case in Fig. 215, (a) pre-operatively, (b) post-operatively. Pre-operatively there was a protrusion of over 1 cm. in the incisor region.

(2) Congenital recession of the jaw

This condition is almost always associated with an overgrowth of maxillary alveolus in the incisor region and a markedly labial articulation of the upper incisors. There is commonly an overgrowth of the nose, for which a reduction may be necessary if the best possible cosmetic appearance is to be obtained.

Cosmetic appearance

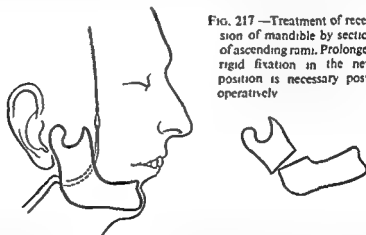


FIG. 217.—Treatment of recession of mandible by section of ascending ramus. Prolonged rigid fixation in the new position is necessary post-operatively.

Operative technique.—The method recommended for the correction of the underslung lower jaw is bilateral resection of the ascending ramus, as for prognathism. The lower jaw is then drawn forward into the required articulation (Figs. 217 and 218). A forward displacement of the whole molar unit can be expected. The jaws are then held together by intermaxillary fixation

with locking bars and wire applied to previously cemented cast-metal splints. The area of contact between the divided portions of the ascending rami is



FIG 218.—Recession of jaw treated by section of ascending ramus and alveolectomy. (Case treated in collaboration with Professor H. R. Fenn.)

narrow. Fixation must be maintained for at least 3 months to ensure consolidation of bone across this gap. It is wise to allow a further period of from 3 to 4 months in elastics or a Schroeder flange before removing the splints.

Later an upper alveolectomy, with extraction of incisors and replacement by a denture, is commonly necessary. Upper
alveolectomy

(3) Correction of grossly asymmetrical occlusion

As in hypoplasia of the first arch, and following osteomyelitis of the mandible, the problem here is one of a grossly disordered occlusion, which may so interfere with mastication as to handicap nutrition, combined with a lower

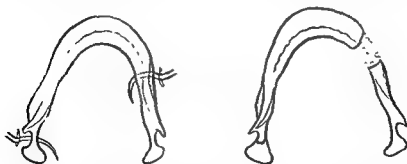


FIG 219.—Treatment of asymmetrical recession of jaw by section, insertion of a bone graft on the short side and section of the condylar neck on the long side.

jaw which is so asymmetrical and receding as to produce a grotesque appearance. The repair problem is to let in bone on the short side of the jaw while the mental prominence is swung forward and into the midline. At the same time it is best to divide the neck of the condyle on the long side, in order that

undue stress is not placed upon the joint and to correct any tendency to recurrence of the deformity which such a stress might cause (Fig. 219).

Operative technique.—At operation, effected through a short external incision below the angle of the short side of the jaw, the mandible is divided at the level



FIG. 220—Asymmetrical recession of mandible following suppurative arthritis of the right temporo-mandibular joint. Treated by section at junction of right ascending and horizontal ramus with insertion there of a bone graft, and section of the left condylar neck to permit of the jaw being drawn forwards and centrally (Case treated in collaboration with Mr W. E. Rix.)



FIG. 221—Side views, pre-operatively and post-operatively, of a case of asymmetrical recession of the mandible, treated by bilateral section, with a bone graft on the short (right) side.

of the angle of the jaw. The neck of the condyle on the long side is then divided by the method of Kostecka, as described in the correction of prognathism. The tooth-bearing segment of the jaw is then swung forward and into the midline where it is fixed in its proper occlusion by intermaxillary fixation with wire and locking bars. The gap on the short side is then filled with a

medullary block of bone. This should be carpentered, shaped and so impacted that it maintains the posterior fragment in its proper position. Fixation of the jaws is maintained for from 2 to 3 months. A further period in elastics or a Schroeder flange may be necessary to prevent the new occlusion from slipping (Figs. 220 and 221).

(4) Correction of congenital open bite

For correction of this deformity it is necessary to divide the horizontal ramus of the jaw on both sides, commonly in the pre-molar region. In order to

*Bilateral
division of
horizontal
ramus*

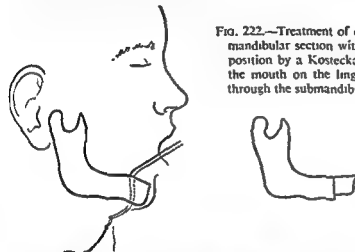


FIG. 222.—Treatment of congenital open bite by mandibular section with a Gigli saw, put into position by a Kostecka seeker passed through the mouth on the lingual side of the jaw out through the submandibular skin.

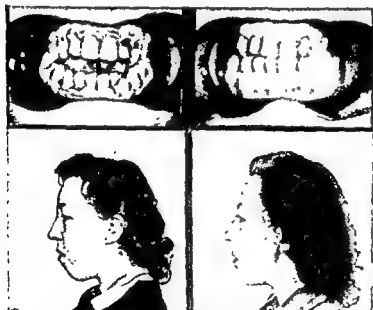


FIG. 223.—Open bite Patient before and after treatment by bilateral mandibular section with the Gigli saw (Case treated in collaboration with Professor W. E. Herbert)

obtain the best possible occlusion of the non-articulating teeth, it may be necessary to resect some bone on one or both sides of the jaws. Such extractions as may be necessary in order to ensure that the line of section does not involve tooth sockets are performed from 4 to 8 weeks before operation.

Operative technique.—At operation a Kostecka seeker is passed at the level of intended section, on the lingual side of the horizontal ramus of the jaw, through the floor of the mouth and out through a stab incision in the sub-mandibular triangle. A Gigli saw is then withdrawn in the usual manner and with this the jaw is divided (Fig. 222). When both sides have been divided a water-tight closure of the mucous membrane is obtained with catgut sutures, and the skin stab wounds are closed with single fine silk sutures. The anterior

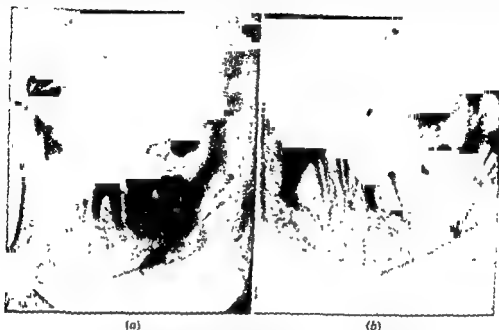


FIG. 224—Skiagraphs of case of open bite in Fig. 223 (a) Before section; (b) after section. Union was present in 6 weeks on the right side and delayed for 3 months on the left side

fragment is then brought up into proper occlusion. Intermaxillary fixation is maintained until union is complete, as demonstrated by clinical tests. It may be possible to release the intermaxillary fixation before firm union is present, provided locking bars are maintained across the fracture sites (Figs. 223 and 224).

(5) Correction of mal-occlusion and gagging due to old fractures

When the mal-occlusion and gagging are too great to be corrected by the grinding of molars or by extractions, a re-fracture of the mal-union, followed by accurate reduction, should be considered. For fractures in the horizontal ramus of the jaw the method of re-fracture which is recommended is that described for the correction of congenital open bite—that is, the fracture site is divided by a Gigli saw.

More commonly the mal-united fracture which causes gagging is in the angle region, and it may be possible in early cases to re-fracture this by closed manipulation. If this is found to be impossible, a Gigli saw, passed by the Kostecka method as for prognathism, can be used to divide the jaw at the site of the fracture. Stainless-steel extra-oral pins of the Rushton-Walker type should be inserted into the posterior ascending ramus fragment in order to maintain the angle in its proper posterior position. The jaws are maintained in intermaxillary fixation until union is clinically sound, which is generally after from 6 to 8 weeks.

3. DEFORMITIES OF THE MAXILLA

In most congenital deformities of the maxilla the cardinal error is its retro-*Retroposition of maxilla* position. The upper teeth are therefore encased within the guard of the lower teeth. The effect, demonstrable with increasing age, is a lack of development of the upper jaw in all dimensions. The final result is a narrow, retroposed, tiny maxilla, with a labial articulation of the teeth showing a variety of types of mal-occlusion. The key to treatment is to bring the alveolar arcades outside their encasement by the lower jaw, at least to an edge-to-edge articulation. This may be accomplished in mild degrees of under-development of the maxilla by orthodontic methods alone. In such cases the essential point in diagnosis is to determine whether it is an overgrowth of the mandible or an undergrowth of the maxilla which is the primary fault. In making this decision, on which treatment will largely depend, cephalograph skiagrams can be of great value.

The operations to be described here for bringing the maxilla forwards and outwards are based upon the work of Gillies. He has shown that the maxilla may be separated from its attachments to the base of the skull and the pterygoid process by appropriate bony sections with the chisel; and that increase in breadth may be obtained by dividing the palate and, if necessary, establishing there a cleft which has later to be filled by an obturator. The alveolar fragments are then brought forwards and outwards and fixed in their new occlusion by intermaxillary fixation of previously applied cast-metal splints supported through rods to a plaster-of-Paris head-cap.

(1) Oxycephaly

The chief facial malformation in this condition is the tiny, narrow, retroposed maxilla; in addition the nose has a grossly sunken bridge in the glabellar region.

Operative technique.—Gillies's method of correction entails: section of the nasal bones and division of the nasal septum from the glabella down to the

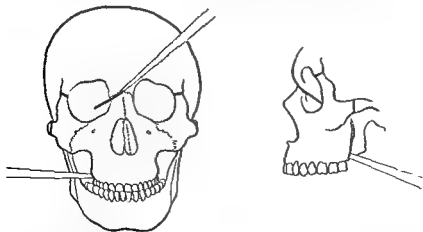


FIG. 225.—Maxillary mobilization. This operation was introduced by Gillies for the late reduction of mal-united fractures; it was subsequently extended by him for use in mobilizing and bringing forward into proper occlusion the narrow and retroposed maxillae seen in cases of cleft palate and oxycephaly.

*Normal
facial contour
restored*

posterior border of the vomer, of the maxilla from the ethmoids, of the angular processes, of the zygomatic arch, and of the tuberosity of the maxilla from the pterygoids (Fig. 225). The maxillae are then drawn forwards into an edge-to-edge articulation, or farther. The upper jaw carries with it the nose and cheeks and produces a normal facial contour. The jaws are fixed together by intermaxillary fixation of previously cemented splints. Bars connect these splints through universal joints to a plaster-of-Paris head-cap. Fixation is maintained until union occurs, that is for from 3 to 6 weeks.

(2) The maxilla in cleft palate

Reviewing the modern position of surgery of the cleft palate Gillies (at the Annual Meeting of the American Association of Plastic Surgeons, Memphis, U.S.A., 1947) has recently re-emphasized that it is not enough to provide normal speech for patients with a cleft palate. The correction of the occlusion and the restoration of normal facial contour have also to be considered. The operative replacement of the maxilla, which is done at 14 to 21 years of age, is essentially that described for oxycephaly; it may be necessary, however, to re-establish a cleft in the hard palate in order to bring the alveolar arcades into edge-to-edge occlusion with the lower jaw and produce a normal occlusion and normal cheek contour. The defect in the hard palate has then to be closed by an obturator.

(3) Mal-occlusion following old fractures of the maxilla

The deformity here comprises a backward displacement of the maxilla together with a tilting of the alveolar arcade, so that gagging, that is open bite, results. This type of mal-occlusion can follow either an alveolar fracture or the higher, middle third type of fracture.

*Rapid
consolidation
of maxilla in
malposition*

The maxilla is noted for the rate at which it consolidates in malposition; even so it is often possible to shake the maxillary fragments loose by manipulation with forceps and to reduce the fracture into normal position as long as from 3 to 6 weeks after the injury. If manipulation at operation is unsuccessful, it may still be possible, during this period, to obtain a reduction by continuous traction. A weight of 2 pounds, applied from a Balkan beam to cast-metal splints on the teeth, together with strong elastic intermaxillary fixation, may produce a reduction. There remain those cases which are too firmly united to be manipulated by such methods. For these the correct procedure is to re-establish the fracture by open operation, using a chisel which separates the alveolar arcade and hard palate from the nasal septum and upper parts of the maxilla. The jaws are then fixed till union in proper occlusion is achieved by intermaxillary fixation of previously cemented cast-metal splints.

4. AFFECTIONS OF THE TEMPORO-MANDIBULAR JOINT

Articulation of the teeth and masticatory function can both be influenced by limitation of movement and by pain in the temporo-mandibular joints. The notes here describe the surgical measures for the relief of painful clicking joints, of osteoarthritis, and of trismus of the temporo-mandibular joints.

(1) Painful clicking joints

Diagnosis

... ..

with reasonable certainty. The functional element in such patients may be marked but conservative treatment is rarely successful.

Operative technique.—The operative approach of choice is the post-auricular one, in which an incision is made from above the ear down towards the mastoid process and carried down to bone. All soft tissues are swept off the bone, the external auditory meatus and the ear are displaced downwards and forwards to expose the outer aspect of the temporo-mandibular joint. The cartilage is removed by incising the capsule, applying haemostats to the cartilage, and freeing it round its margin by dissection with scissors. The skin incision is closed in one or two layers and active movements of the jaw, employing chewing-gum and a wedge, are practised from the immediate post-operative period.

Post-auricular approach

(2) Painful osteoarthritis

A certain number of cases of osteoarthritis respond to conservative methods of treatment, such as the injection of Novocain, lactic acid or iodized oils. In other cases in which constant pain and crepitations are present in the joint it is an old cartilage injury which is the cause, and removal of this may result in a considerable lessening of pain and an improved range of movement. In a third group, of persistent and long-standing cases, the pain may be constant and disabling, and associated with severe trismus. For such cases more radical measures are necessary.

Conservative treatment

More radical measures

Operative technique.—For these patients, relief may be provided by an excision of the head and neck of the condyle. Bilateral removal of the condylar heads, however, can result in intractable gagging. It is therefore often wiser in bilateral severe osteoarthritis of the temporo-mandibular joints to remove a wedge of bone below the neck of the condyle across the whole ascending ramus of the jaw above the inferior dental foramen (Fig. 226). This is done

Risk of intractable gagging



FIG. 226.—Mandibular resection for severe trismus and severe osteoarthritis of the temporo-mandibular joints. Through a small incision Gigli saws are placed in position by a Kostecka seeker.



The ascending ramus is divided at two points and the enclosed piece of bone removed by dissection to leave a false joint which is painless and mobile.

through a low infralateral approach. The incision is from the top of the mastoid process 1-2 inches towards the angle of the jaw. The posterior border of the ascending ramus and the neck of the condyle are cleared. The mandible is divided above the level of the inferior dental foramen by a Gigli saw put in position by a Kostecka seeker. Division of the ascending ramus here permits traction on the upper fragment, which is divided with bone-cutting forceps below the neck of the condyle; alternatively the condyle is removed in one piece with the upper ascending ramus of the jaw. Post-operatively the jaws

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ORTHODONTICS—SURGERY OF

are kept gagged open continuously for 2 weeks, if section is below the condyle (Fig. 227). Subsequently the gag is worn every night for from 6 months to a year while active movements are practised. If the condyle has been removed



FIG. 227.—Severe osteoarthritis with clicking and pain of the temporomandibular joint. Treated by resection of ascending ramus as in Fig 226, and post-operative gag. Operation gave a slight increase in the range of movement of the jaw and relief of pain on the affected side. (Case treated in collaboration with Mr. E. G. Dalling)

[illegible]

(3) Bony ankylosis

(3) **Bony ankylosis**—The most effective relief of bony ankylosis is provided by the Esmarch type of operation. The removal of a wedge of bone, 1 inch in vertical depth from the ascending ramus, is facilitated by the use of the Kostecka seeker. Through a 1-inch incision passing downwards from the mastoid process the seeker is passed so that the mandible is divided just above the level of the horizontal ramus of the jaw. It is then passed again so as to make a second line of section just below the tip of the coronoid. The free intermediate fragment is then taken with bone-holding forceps. Its soft-tissue attachments are peeled from it as it is retracted through the incision. In this dissection the inferior dental nerve and artery are divided. The incision is then closed in two layers. The jaws are kept gagged open for 2 weeks. Subsequently the gag is worn at night for from 6 to 9 months, and active movements are practised.

5. OPERATIONS ON THE SOFT TISSUES

(1) Lip bands

(1) Lip bands
A lip band passing from the centre of the upper lip to the alveolus between the upper incisors is often associated with wide separation and labial

articulation of these two teeth. The band should be corrected by a "Z" plastic operation. At the same operation it is necessary to remove a wedge of soft tissue and bone from between the roots of the incisors to allow these teeth to come to lie together in proper articulation.

(2) Bulky tongues

An excessively large tongue, particularly in its anterior portion, can be a cause of open bite and of labial articulation of upper or lower teeth. When this is the case a central anterior wedge should be resected from the tongue. Removal of such a wedge shortens the tongue and narrows it anteriorly. It may also be necessary to give patients instruction regarding proper tongue movements so that they do not continue to practise such faulty movements as rolling the tongue against the incisors.

(3) Epithelial inlay for edentulous patients

These notes concern the provision of dentures for elderly edentulous patients. The alveolar ridge decreases in depth with age. It is therefore often very difficult to fit a stable denture for the elderly patient because of the shallowness of the buccal sulcus. This sulcus may very readily be deepened to the necessary amount by provision of an epithelial inlay. The operation may be done under local anaesthesia.

Operative technique.—A sheet of thin split skin, about 6 inches by 4 inches in area, is cut from the inner arm. The buccal sulcus is deepened to the necessary extent by incision from molar to molar region. A gutta-percha mould is then fitted into the deepened sulcus and the denture impacted into it. The mould and denture are then withdrawn and the free graft is spread over the mould, the raw surface outwards. The graft may be fixed to the mould by Benzo Mastic. The mould and both dentures are then replaced in the mouth and the chin is supported by a crêpe-bandage strap for 10 days. The deepened epithelial sulcus should be ready for a new denture in 6 weeks.

[References to other titles are given under Orthodontics—Surgery of, in the Index Volume.]

ORTHOPTIC TRAINING

BY MARY A. PUGH, M.R.C.S., L.R.C.P.
MEDICAL OFFICER, ORTHOPTICS DEPARTMENT, CENTRAL BRANCH, MOORFIELDS,
WESTMINSTER AND CENTRAL EYE HOSPITAL, LONDON

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253.] Orthoptic treatment aims at restoring a squinting eye to its correct position and establishing normal binocular vision between the two eyes, so that the patient shall have the advantage of stereoscopic vision together with a normal appearance.

1. DEFINITION OF ORTHOPTICS

2. AETIOLOGY OF STRABISMUS

The most common cause of a squint is an error of refraction, such as a high degree of hypermetropia or anisometropia with a marked difference between the refraction of the two eyes. A defect in the capacity for fusing the images seen by each eye may arise during the first 4 or 5 years of a patient's life. Unless normal fusion is developed, there is no stimulus to hold the eyes straight. Psychological traumas, such as fear or shock, may cause the sudden onset of a squint even in adult life. Imitative squints occur in young patients. A certain number of children, either from birth injury or maldevelopment, are born with a defect which may manifest itself as a squint. The sight of one eye may be irretrievably poor, or one or more muscles may be defective.

3. PHYSIOLOGY

In focusing both eyes together on to an object the convergence accommodation-link enables the extrinsic muscles to work with the intrinsic ciliary muscle, so that the object is focused sharply at the same time as each visual axis is converged on the object, which is thus seen clearly and with each eye simultaneously.

4. PATHOLOGY

From the above paragraph it is obvious that in a long-sighted eye extra accommodation is necessary to bring an object to a sharp focus; but extra

Emmetropia

Hypermetropia

accommodation means extra convergence. Although some patients manage to adjust to this abnormal linkage between the two sets of muscles, many are unable to do so and the over-convergence soon develops into a convergent squint. Whenever the refraction of the two eyes is dissimilar, or there is unequal myopia, corresponding difficulties arise.

When one eye is deviated from its normal position diplopia is likely to occur. This symptom rarely remains constant, and the patient learns either to suppress an eye or to correspond the retinal points of each retina in an abnormal way in order that he may be able to make the two images coincide. If one eye is always suppressed in a child under 5 years of age, the danger that good vision will fail to develop in the eye is considerable. *Suppression*
Anislopia

5. CLINICAL PICTURE

The average age of onset of the squint is about 2 or 3 years, although the condition may be present from birth. If the onset is later than 5 years it is probably not a straightforward case. *Age of onset*

In a monocular strabismus one eye squints while the other eye is constantly used. The weaker eye may converge or diverge if the internal and external recti are not co-ordinating, it may deviate up or down if the superior or inferior recti are affected, or it may take up an oblique position combining a vertical with a lateral axis if the oblique muscles are affected. *Monocular squints*

An alternating squint is one in which the patient can use either eye easily, and the vision in each eye is therefore well developed. The direction of the deviation may be as in a monocular squint. *Alternating squints*

6. SPECIAL AIDS TO DIAGNOSIS

Refraction, determined under atropine, will give the most useful information. High hypermetropia, marked anisometropia or astigmatism, and unequal myopia are the common errors found. The correction of these errors by suitable spectacles is the first essential step before anything else can be done. *Refraction*

The vision in each eye, with its correcting lens, must be assessed. If the vision of one eye is below normal, the strong eye must be covered with a patch until normal vision has developed in the weaker eye. Illiterate children can be tested with charts illustrating animals of various sizes, or with an E device which turns round to point right, left, up or down. *Vision with correcting lenses*

Binocular vision is tested by various instruments, which dissociate the two eyes and at the same time present an object for each eye to see. The patient says whether he sees with one eye or two eyes at the same time, and how the two objects, if seen at the same time, are related to each other in space. *Binocular vision*

The most simple correction is a patch on the middle of the spectacle. The patient is

A stereoscope is valuable to assess suppression, fusion and amount of amplitude and stereoscopic vision, but is not of much use in deciding whether the retinal corresponding points are normal or whether a new abnormal correspondence has developed. *Stereoscope*

Amblyoscopes Amblyoscopes can measure complicated deviations and, as the pictures can be placed to fall on the macula of each eye simultaneously, the answers will give either:

- (1) *Suppression* of one or either eye, constant or intermittent.
- (2) *Simultaneous macular perception* = Grade I binocular vision = dissimilar pictures accepted together, such as a bird seen in a cage.
- (3) *Fusion of plano pictures* with some range of fusion = Grade II binocular vision.
- (4) *Stereoscopic vision* = Grade III binocular vision, in which slightly dissimilar pictures can be fused and the resulting differences of the positions of the objects in space can be appreciated.
- (5) *Abnormal retinal correspondence.*

7. DIFFERENTIAL DIAGNOSIS

Absent or defective muscles

Babies may be born with external rectus muscles absent or, more rarely, with developmental defects in the separate arrangements of one or more muscles. Forceps or difficult delivery can give rise to trauma, which may leave a muscle paralysed or weakened.

Central nervous system

A brain tumour, haemorrhage or aneurysm, or any trauma to the third, fourth or sixth cranial nerve centre can cause paresis or paralysis of an ocular muscle and, in consequence, deviation of an eye.

Diseases of the central nervous system, such as disseminated sclerosis or encephalitis, may cause diplopia as a result of inco-ordination of the extrinsic eye muscles. Exophthalmic ophthalmoplegia is easy to recognize when the condition is established, but in the early stages of some types of thyroid trouble the main symptom is diplopia with only slight indications of possible hyperthyroidism.

Peripheral nerve trauma

Peripheral lesions which affect the nerves or muscles must be excluded. Local new growths, such as a dermoid cyst in the orbit, can obviously interfere with the normal position of the globe.

8. PROGNOSIS

Prognosis is good provided that:

- (1) Amblyopia is avoided by covering the strong eye as soon as possible after the eye has deviated.
- (2) Correcting lenses are worn constantly whenever there is any appreciable error of refraction.
- (3) Normal binocular vision is developed as soon as the child is able to co-operate. Small amblyoscopes and stereoscopes at home are useful when the patient lives far from a medical centre.
- (4) Surgery is performed when necessary.

9. INDICATIONS FOR SURGICAL INTERVENTION

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The optimal age for operation must be decided according to the type of squint and the type of patient.

If spectacles, occlusion and exercises have not proved successful measures for getting the eyes straight by 7 years of age, then, in the average case, they

should be put straight by surgery. At this age the patient is old enough to co-operate in the post-operative treatment and exercises.

If the angle of squint is very big and mechanically defeats any effort at treatment, it may be advisable to operate much earlier, say at 2 or 3 years of age. A further operation for a final result can be undertaken, if needed, when the child is about 7 years of age. On the other hand, some patients with psychological difficulties may not be ready for surgery by the age of 7 years. Often they will not co-operate and, in fact, until they have reached their teens, do not want to lose the squint. *Degree of deviation*

10. PRE-OPERATIVE TECHNIQUE OF ORTHOPTICS

The causative factors have been removed as far as possible. Exercises have been given to break down suppression and abnormal correspondence and to develop normal fusion and stereoscopic vision. Careful measurement of the angle of the squint with and without glasses, together with the measurement of the degree of binocular vision present, will allow the surgeon to decide on his exact surgical technique before operation. This means that the patient can be given a general anaesthetic if it is thought advisable.

11. POST-OPERATIVE CARE UP TO CONVALESCENCE

(1) Immediate post-operative care

Both eyes are covered for approximately 7 days after the operation. If there is any tendency for the eyes to converge, atropine, 1 per cent, is used in both eyes twice a day. On the seventh day, if the eyes are straight and have Grade I, II or III binocular vision, the patient is allowed to use both eyes together, with spectacles for distance vision. No reading, sewing or near work is allowed for about a month.

(2) Exercises with the stereoscope

Exercises with a simple stereoscope will help to keep the eyes steady and to make them use normal binocular vision in the usual routine of the day. The exercises may be done 2 or 3 times a day for 20 minutes at a time.

(3) Continued occlusion

When fusion has not been developed before operation, post-operative occlusion may have to be continued for some months until the tendency for the eye to recover its original squint has disappeared. Diplopia in these cases may last from 6 to 12 months and, in occasional instances, even longer.

12. RESULTS OF TREATMENT

With care excellent results may be obtained, but continued care must be taken during the growing years of the patient. For instance, whenever the primary cause was a high error of refraction this will remain a potential cause for a recurrence of the condition, unless correcting lenses are worn. Again, a patient whose binocular vision has once been wrong may relapse, and a squint may occur unless both eyes are used together. If the eyes have remained straight for a year after the operation, reasonable care should stabilize the result.

ORTHOPTIC TRAINING

BIBLIOGRAPHY

- Bielschowsky, A. (1922). *Stellungsanomalien und Beweglichkeitsstörungen der Augen, Nystagmus, Störungen der Pupillenreaktion. Exophthalmus und Enophthalmus, Störungen des Gesichtsfeldes. Zerebrale und psychogene Störungen* Leipzig; Thieme.
- Cantonnet, A., Filliozat, J., and Fombeure, G. (1938). *Strabismus. Its Re-education* The Physiology and Pathology of Binocular Vision, 2nd ed. Trans. by Coque, M. Vol. 2. London; Kimpton.
- Duke-Elder, in the Treatment of Squint, Guibor, G. P. (1925), Lyle, K., and Jackson, Sylvia (1940). 2nd ed London; Lewis.
- Peter, L. C. (1936). In *The Eye and its Diseases*. Ed. by Berens, C. Philadelphia; Saunders
- Pugh, M. A. (1936). *Squint Training*. London; Oxford University Press.
- Sattler, C. H. (1929) *Int ophthal Congr.* (Amsterdam). Vol. 1, p. 116.
- Travers, T. AB (1936). *Brit J. Ophthal.*, Supplement No. 7.
- Wells, D. W. (1928) *The Stereoscope in Ophthalmology*, 4th ed. Boston; Mahady.
- Worth's Squint, or the Binocular Reflexes and the Treatment of Strabismus*, 7th ed (1939). Ed by Chavasse, F B London; Baillière, Tindall & Cox.
- [References to other titles are given under Orthoptic Training in the Index Volume.]

OVARY

BY WILFRED SHAW, M.D., F.R.C.S., F.R.C.O.G.

SURGEON-IN-CHARGE, GYNAECOLOGICAL AND OBSTETRICAL DEPARTMENT,
ST. BARTHOLOMEW'S HOSPITAL, LONDON

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1. INTRODUCTION

254.] In the surgery of the ovary much depends upon the recognition of the pathological conditions which are found at operation, and great judgement may be required to decide upon the surgical procedure to be adopted.

2. CLASSIFICATION OF PATHOLOGICAL CONDITIONS

A simple method of classifying the pathological conditions of the ovary is to regard them as falling into one of the following groups:

- (1) Neoplasms
 - (a) Innocent
 - (b) Malignant
- (2) Fimbrial cysts
- (3) Chocolate cysts of the ovaries combined with other forms of pelvic endometriosis
- (4) Inflammations of the ovary
- (5) Sclerocystic ovary
- (6) Physiological cysts

(1) Neoplasms

(a) *Innocent*

The commonest form of ovarian neoplasm is a cystoma or a cystadenoma, and it takes the form either of a unilocular cyst or, more commonly, of a multilocular cystadenoma filled with pseudomucin. In the average case the tumour is freely movable and free of adhesions, and its surgical removal offers little difficulty. In some cases the ovarian cyst is papillomatous and the papillomas may be either intra-cystic or scattered over the surface of the tumour. In advanced cases the surface papillomas may be diffused over the pelvic peritoneum so that the tumour, although pathologically innocent, is clinically malignant. It should be remembered that malignant changes can be demonstrated on microscopical examination in 6 per cent of all cases of seemingly innocent ovarian tumours.

Dermoid cysts rarely attain a large size. Their consistency is peculiar and quite frequently the tumour does not regain its original shape if the wall has been indented, because of the oily and waxy nature of its contents.

Solid ovarian fibromas are connective-tissue tumours which are almost always free of adhesions. They may attain a large size and are often associated with ascites and sometimes with hydrothorax.

(b) *Malignant*

At least 25 per cent of all ovarian tumours are malignant. Moreover, at least 20 per cent of all malignant tumours of the ovary are secondary growths from primary growths elsewhere in the body, particularly from carcinoma of the stomach and malignant tumours of the large bowel and breast. Sarcomas are relatively uncommon. The rare granulosa-cell tumour, the dysgerminoma and the virilizing tumours such as the arrhenoblastoma almost always have a low degree of malignancy.

(2) Fimbrial cysts

These tumours develop in the outer part of the broad ligament, separate from the ovary; the ovarian fimbria is characteristically spread out over the outer part of the tumour. The tumours should be regarded as neoplasms and are comparable to serous cystomas and pseudomucinous cystadenomas. They are not retention cysts. They can be easily identified at operation because of their anatomical relations.

(3) Chocolate cysts and endometriosis

These are seen far more frequently than in previous years. In the typical case, both ovaries are moderately enlarged and the tunica albuginea is thickened and white. The tumours are densely adherent to the back of the broad ligament, to the peritoneum of Douglas's pouch and to the posterior surface of the uterus. Scattered dark-red areas of endometriosis are distributed over the back of the uterus and the pelvic peritoneum. Almost always the cyst ruptures as it is being excavated from the pelvis and dark chocolate-like fluid is discharged into the peritoneal cavity. The condition is caused by heterotopic endometrial proliferations which are probably induced by hormonal action, and the tumours should not be regarded as malignant. It is doubtful

lated
is

Innocent
papillomatous
cysts

Dermoid
cysts

whether malignant change has ever been demonstrated in well-established chocolate cysts of this kind.

(4) Inflammations of the ovary

The ovaries are usually infected as the result of the upward spread of the gonococcus or of pyogenic organisms from the uterus by way of the Fallopian tubes. As the ovaries lie symmetrically with respect to the uterus, inflammation is usually bilateral. In tuberculous infections of the ovaries and tubes, the bacteria reach the ovaries either by way of the blood-stream or by implantation from the peritoneal cavity as the result of the rupture of a caseous tuberculous mesenteric gland.

In all infections of the ovary the Fallopian tube is infected as well. A pyosalpinx or a hydrosalpinx may be a more marked feature of the case than an ovarian abscess or oöphoritis. Sometimes a pyosalpinx communicates with an ovarian abscess to produce a tubo-ovarian abscess. In old-standing cases after the initial infection has died down adhesions form around the ovaries and tubes and fix the ovaries to the back of the uterus.

*Pyosalpinx
and
hydrosalpinx*

(5) Sclerocystic ovaries

Patients sometimes complain of ovarian pain, and in these cases the ovaries may be found at operation to have sclerosed tunics. Often the ovaries are much enlarged, each with a thickened, firm, greyish-white tunica. At other times pain in the right iliac fossa may be complained of, and it is important when the appendix is being explored to know whether the appearance of the ovaries suggests that pain emanates from the ovary and not from the appendix. If the ovary is sclerosed and enlarged, it may be advisable to excise the cortex.

*Ovarian
pain*

(6) Physiological cysts

Corpus luteum cysts should be regarded as physiological in that they retrogress if left alone. They are encountered particularly in early pregnancy and with ectopic gestation. They are characterized by their yellow contents and their thin walls. With hydatidiform mole and chorionic epithelioma, multiple follicular cysts are present as well. The ovary is studded with cysts of about half an inch in diameter. Such appearances are pathognomonic of hydatidiform mole and chorionic epithelioma. Sometimes cysts of this type undergo torsion.

3. CLINICAL PICTURE

(i) *Neoplasms*.—Ovarian neoplasms—even mammoth ovarian cysts—rarely affect the menstrual functions. Patients usually complain either of pressure symptoms or of an abdominal swelling. In innocent tumours the surface is smooth, the percussion note is dull and, except in cases of fibromas of the ovary, there is no ascites. The diagnosis of malignant change can usually be made before operation. Most malignant ovarian tumours are bilateral and the tumours are mainly solid. Ascites is nearly always present. Secondary deposits can be palpated, both in Douglas's pouch through the posterior vaginal wall and in the omentum at the level of the umbilicus. Wasting is not a certain sign of malignancy, for it may develop to an extreme degree even with innocent tumours. If bilateral ovarian tumours are discovered clinically, the

Ascites

chances are very much in favour of the tumours being malignant. If there are widespread metastases in Douglas's pouch and in the omentum surgical intervention is contra-indicated, for most patients go downhill rapidly even after a simple laparotomy.

(ii) *Chocolate cysts and pelvic endometriosis*.—The patients in whom these occur are always in the child-bearing period of life. They complain of dysmenorrhoea, dyspareunia, sterility, menorrhagia and backache. Emergency cases are seen from time to time as the result of rupture, when the chocolate-like fluid is discharged into the peritoneal cavity.

(iii) *Inflammations of the ovaries and tubes*.—The majority of cases follow acute gonorrhoea, septic abortion and puerperal sepsis, while tuberculous cases are more chronic. Abdominal pain following acute vaginal discharge or a septic abortion should always make one suspect the presence of salpingo-oophoritis, and bimanual examination should enable the enlarged appendages to be palpated. Widespread peritonitis or pelvic abscess may complicate the severe cases. In tuberculous salpingo-oophoritis, the main complaints are abdominal pain, sterility and backache, together with menorrhagia.

(iv) *Sclerocystic disease*.—In sclerocystic disease and with painful ovaries, the main complaint is dull pain located in the lower abdomen, but the ovaries should be palpated bimanually, and only if compression of the ovary elicits the pain of which the patient complains should the diagnosis of "ovarian pain" be made.

4. OPERATIVE FINDINGS

The first step is to establish the nature of the tumour, and this can be done only by identifying the relations of the tumour by means of the attachment of the ovarian ligament to the uterus, by the tumour's connexion to the back of the broad ligament and its proximity to the Fallopian tube. In mammoth tumours it may first be necessary either to tap the tumour or to remove it from the abdomen before its exact relations can be identified. The anatomical relations of a fimbrial cyst must be determined, for such tumours can be shelled out of the broad ligament quite easily, the ovary and Fallopian tube being conserved. If the tumours are bilateral and solid, they are probably malignant; if they are fixed as well, they are almost certainly malignant. If there is well-marked ascites with metastases, further intervention is unjustifiable.

Sometimes innocent tumours burrow extra-peritoneally, and in some cases membranous adhesions may fix the tumour to the intestines and peritoneum, so that the complete surgical removal of the intact cyst may be impossible. Nevertheless, an effort should be made to shell out the tumour, and this may be possible if the correct layer of cleavage is found. The ureter may lie in close proximity to tumours of this kind, so that great care must be taken in the dissection along the lateral wall of the true pelvis.

In chocolate cysts and pelvic endometriosis very great difficulty is experienced in separating the lower pole of the tumour from the peritoneum of Douglas's pouch, from the posterior surface of the uterus and from the sigmoid colon. There is no layer of cleavage and the tumour must be cut away from its adhesions with scissors. The infiltration is always best marked in the vicinity of the peritoneum of Douglas's pouch.

Rupture of cyst

Identification of fimbrial cyst

chocolate cysts

With inflammations of the ovaries and tubes the adhesions are not necessarily particularly dense, except in tuberculous cases; it is usually possible to separate the ovary and the inflamed tube digitally. The adhesions are most marked towards the lower pole of the ovary in the region of Douglas's pouch, and it is best to start the separation from below and to work upwards, the hand being insinuated posteriorly between the tumour and the posterior wall of the true pelvis. Tuberculous salpingo-oöphoritis should be capable of recognition by inspection. In simple cases miliary tubercles are scattered over the swelling; dense membranous adhesions are also characteristic of tuberculous cases. With hydrosalpinx, although membranous adhesions are present, the wall of the tube is thin and there is no difficulty in distinguishing such cases from those of tuberculosis. If it has been decided to remove the ovaries and tubes in tuberculous cases, great care must be taken to avoid damage to the bowel, because of the risk either of peritonitis or of faecal fistula.

Inflammations

Tuberculous infections

Decision as to whether both ovaries should be removed

If the patient is of menopausal age, the opposite ovary should be removed as a routine, irrespective of whether it contains cysts or not, for the post-menopausal ovary serves no function. If one ovary has developed a tumour, whether innocent or malignant, the risk of subsequent involvement of the other ovary in tumour formation is relatively high. Moreover, even seemingly innocent tumours are found to be malignant in 6 per cent of cases. If the tumours are bilateral and solid, have the appearance of malignant tumours and yet can be removed without much difficulty, statistics show that the best results are obtained if the uterus is removed as well; therefore a subtotal hysterectomy should be performed as a routine.

Unilateral tumours

Bilateral tumours

If the patient is in the child-bearing period of life and has bilateral innocent tumours, an effort should be made to shell out the tumours by the operation of ovarian cystectomy, so that some functioning ovarian tissue is retained. Such cases are by no means infrequent, particularly in the case of bilateral dermoid cysts, and these tumours can be shelled out without much difficulty. Experience shows that it is almost unknown for further tumours to develop in the ovarian tissue which has been retained.

Clearly, much experience is required to determine the nature of the tumours by simple inspection at operation. With chocolate cysts, the position is one of great difficulty and complexity. Chocolate cysts and pelvic endometriosis tend to recur after the removal of a unilateral chocolate cyst. If the condition is bilateral, with well-marked pelvic endometriosis and an enlarged uterus, in spite of the age of the patient it is best to remove both ovaries and both Fallopian tubes, together with the uterus. If the chocolate cyst is unilateral it should be removed, but if the uterus is enlarged and there is pelvic endometriosis as well, the patient will not be cured of her symptoms. The principle of treatment is to remove both ovaries and the uterus in advanced cases; yet if healthy ovarian tissue can be conserved, it will prevent the patient from developing symptoms of an artificial menopause, although the uterus may have to be sacrificed. Sterility is invariable if chocolate cysts and endometriosis are far advanced, so that the question of conserving the uterus is not so important as it may seem to be at the time of operation.

Chocolate cysts

Sterility

Sclerocystic ovaries

With sclerocystic ovaries and ovarian pain the condition is often bilateral, and as it may be necessary to excise the cortex of both ovaries, the opposite ovary should always be carefully inspected in these cases.

5. OPERATION

(1) Tapping

Tapping should be avoided if there is any suspicion that the tumour is malignant. Occasionally, elderly patients with coronary disease are found to have innocent ovarian cysts, which can be treated by tapping without subjecting the patient to a laparotomy. Such cases are few, for it is possible to remove very large ovarian tumours under local anaesthesia.

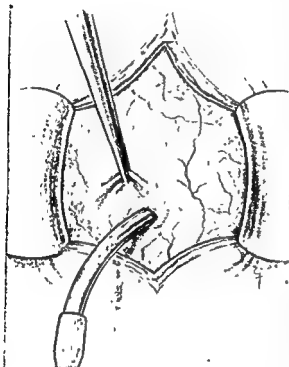


FIG. 228.—Method of tapping an ovarian cyst. A relatively small incision is required and a trocar and cannula are inserted into the tumour.

When dealing with mammoth tumours, tapping reduces the size of the tumour and thus simplifies the excision. It is possible to remove very large ovarian tumours through a small abdominal incision if the tumour is tapped after the peritoneum has been opened (Fig. 228). Mammoth tumours are only rarely malignant and tapping is justifiable in that the patient is spared a large abdominal incision. Much depends upon the surgeon being able to detect the nature of the tumour after the abdomen has been opened. If the wall of the cyst is thin and pearly white, the tumour is almost certainly of the pseudo-mucinous type,

whereas if the tumour has solid areas or is thick-walled, it should be regarded as malignant and, in such cases, tapping must be avoided.

(2) Incision and surgical technique

A paramedian incision is customary, and if the surgeon works from the left side of the patient, he usually makes a left paramedian incision which can be extended upwards if necessary. With small tumours, the Pfannenstiel transverse incision gives a satisfactory exposure and the scar is subsequently hidden by the pubic hair. The presence of ascites suggests that the tumour is malignant, almost the only exception being an innocent fibroma of the ovary. Similarly, secondary deposits scattered over the omentum indicate that the tumour is malignant and it may not be justifiable to proceed further. In most cases the ovarian tumour is freely movable, without adhesions, and can be extracted from the abdominal cavity by inserting the right hand behind the

Pfannenstiel incision

tumour. In this way tension is applied to the pedicle, so that the structures forming it are well defined. On the lateral side of the pedicle are the ovarian vessels, while on the medial side are the ovarian ligament with the Fallopian tube usually running over the surface of the cyst. Between the two margins lies the mesovarium which is relatively bloodless (Fig. 229). Three main clamps are placed on the pedicle, one to enclose the ovarian vessels, the second to contain the ovarian ligament and the base of the Fallopian tube, while the third clamp is placed over the mesovarium (Fig. 230). The clamps should be slightly curved and heavy, and their grooves should run parallel to their length, instead of transversely. The pampiniform plexus of veins may be enormously dilated and, to prevent retraction of the vessels extra-peritoneally, an extra clamp should be placed over the lateral part of the pedicle before the tumour is removed. One of the complications of the operation is for

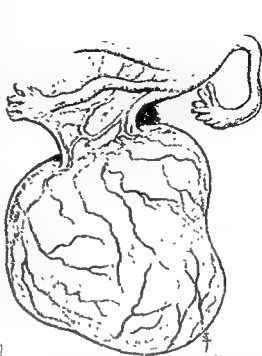


FIG. 229.—An ovarian cyst with its pedicle. The pedicle consists of the ovarian ligament on the mesial side together with the mesovarium attached to the back of the broad ligament in the middle. On the outer side is the infundibulo-pelvic fold which extends outwards to the wall of the pelvis.

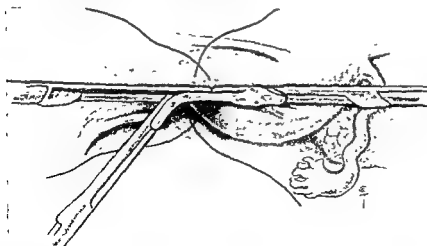


FIG. 230—Following the removal of the cyst, the vessels of the infundibulo-pelvic fold.

the vessels to retract away from the clamp and to bleed extra-peritoneally. Considerable difficulty may then be experienced in dealing with haemorrhage of this type. Thirty-day chromicized catgut No. 2 should be used as ligature material, and at least two separate ligatures should be placed over each part of the pedicle. With left-sided tumours it may not be necessary to cover the raw area with peritoneum, for the sigmoid colon drops down and covers

*Coincident
tumours*

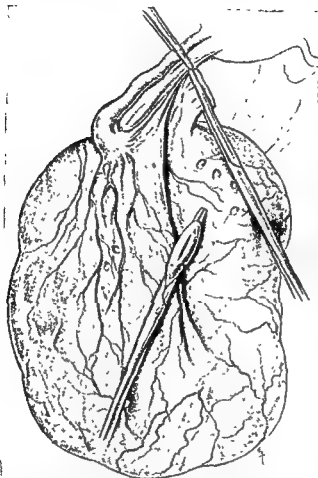


FIG. 231 —The removal of an ovarian tumour which has been tapped. The lower clamp closes the opening through which the tumour has been tapped. The upper clamp includes the Fallopian tube and the ovarian ligament, while the middle clamp in this illustration includes the large vessels of the infundibulo-pelvic fold.

ovarian tumour in the vicinity of the ovarian ligament are controlled as soon as the uterine vessels have been divided between clamps (Fig. 231). In some cases of chocolate cysts, the uterus and the affected ovary on the opposite side must be removed. The procedure is exactly the same as with subtotal hysterectomy.

(3) Ovarian cystectomy

It is well known that most innocent ovarian cysts can be shelled out of the ovary without much difficulty, and it may be in the patient's interest for some

the pedicle, but with right-sided tumours an effort should be made to cover the pedicle with peritoneum if the raw surface is at all extensive. The peritoneal covering can be obtained by mobilizing the peritoneum lateral to and above the pedicle with the help, if necessary, of a longitudinal incision placed just above the pedicle. A leaf of peritoneum can then be drawn down over the raw surface.

If the uterus contains myomas or if there are any other indications for removing the uterus, the ovarian cyst and the uterus should be removed together. The lateral margin of the pedicle should be cut through between clamps. Next, the round ligament should be divided between clamps and the broad ligament exposed in the usual way until the uterine vessels on that side are exposed. The branches of the uterine artery which ultimately pass to the

normal ovarian tissue to be retained. The operation can often be performed with great ease if the correct layer of cleavage is identified. The operation is serviceable for young patients with such innocent tumours as dermoid cysts and simple cystomas, and it is especially indicated in cases of bilateral innocent cysts arising in young women. The term, ovarian cystectomy, has been advocated by Bonney for this operation in contrast to ovariectomy which refers to the removal of the tumour and any ovarian tissue attached to it. Ovarian cystectomy is contra-indicated if there is any possibility that the tumour is malignant.

Innocent papillomatous ovarian cysts sometimes have surface papillomas scattered over the peritoneal surface. It is important to distinguish between innocent papillomas of this type and the dense, friable papillomas of malignant tumours. The innocent papillomas are either warty in appearance or delicate villous-like tumours. If the pelvic peritoneum is studded with such papillomas it is essential that both ovaries should be excised and as many as possible of the papillomas removed from the peritoneum. The ultimate prognosis is always indefinite and there is no reason to believe that such papillomas disappear after radiotherapy. Very great experience is required before a surgeon can judge whether such papillomas are innocent or malignant.

(4) Pseudomyxoma of peritoneum

Pseudomyxoma of the peritoneum is a very rare complication. Although its association with a pseudomucinous tumour of the ovary is well known, the condition is often combined with a mucocele of the appendix or with a carcinoma of the large intestine. The pathology of the condition is little understood, and it is not believed at the present day that the condition always develops as the result of a rupture of a pseudomucinous tumour of the ovary. In surgical practice the ovarian cyst should be removed and both the appendix and the large intestine examined carefully.

(5) Adhesions

Many types of adhesions are found with ovarian tumours. In the simplest form adhesions result from torsion when, as the result of inflammatory reaction from venous congestion, the omentum and intestines become adherent to the tumour. Not infrequently the tumour becomes adherent to the peritoneum of the anterior abdominal wall. In surgical practice these adhesions are usually separated without much difficulty, but those which extend to the anterior abdominal wall call for great care lest the tumour is opened during the abdominal incision. Extreme care may have to be taken to find the correct layer of cleavage to separate the tumour from the peritoneum. The adhesions which form around chocolate cysts are particularly dense, especially those to the rectum and sigmoid colon. The adhesions can rarely be separated with the fingers and must be cut through with scissors. Great care must be taken to avoid injury to the bowel. Moreover, troublesome haemorrhage occurs from the raw surfaces which are left. These must be dealt with either by picking up the bleeding-points or by the use of hot compresses, or of fibrin foam and Thrombin. Sometimes, as the result of puerperal sepsis or of gonococcal infections, dense adhesions form around the ovarian tumours so that

OVARY

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the tumours become fixed in the pelvis and, at first glance, may seem to be extra-peritoneal. Much patience may be necessary to find the correct layer of cleavage and to separate the adhesions, but complete removal of the tumour is usually possible. Very rarely thin-walled ovarian cysts are surrounded by thin membranous adhesions and the posterior layer of the cyst wall may be fused to the posterior parietal peritoneum to such a degree that removal of the tumour is impossible. Fortunately such cases are rare and treatment must consist of marsupialization. It is well known that the tumour can usually be removed without much difficulty after an interval of about 6 months. Extra-peritoneal development of an ovarian cyst is a very rare complication, although cases are seen from time to time. The tumour can be shelled out without much difficulty, but especial care must be taken to identify the ureter and to avoid damage to it during the removal of the cyst.

*Malignant
tumours*

With malignant ovarian tumours the adhesions are the result of infiltration of the surrounding organs by the growth and are of the type which cannot be divided or separated.

6. EMERGENCY SURGERY OF OVARIAN TUMOURS

(1) Torsion of the pedicle

Torsion of the pedicle may cause the symptoms of an acute abdomen and demand immediate surgical intervention. The cases are far less frequent than in past years. The dominant symptom is severe abdominal pain associated with the symptoms and signs of peritoneal irritation. It is only rarely that there is difficulty in diagnosis. The tumour is of necessity mobile and its excision presents no difficulty. Torsion of an ovarian cyst during pregnancy is seen very rarely, but the tumour must be removed.

*Severe
abdominal
pain*

(2) Rupture of ovarian cyst

Rupture of an ovarian cyst is usually spontaneous and is encountered in rapidly growing tumours. It is rare for severe abdominal symptoms to develop. Rupture as the result of trauma is rarer still. From time to time acute cases are met with at puberty. Sometimes a large follicular cyst develops in the ovary with haemorrhage into the wall or with rupture of the cyst into the peritoneal cavity, associated with intra-abdominal haemorrhage. Laparotomy is required and it is usually necessary to remove the whole ovary. Rupture of a chocolate cyst is seen not infrequently. An immediate laparotomy is required and the treatment follows the lines already outlined.

7. MANAGEMENT OF CORPUS LUTEUM CYST

The physiological corpus luteum cyst is found fairly commonly in the early weeks of pregnancy and retrogresses spontaneously. The diagnosis should be made on clinical ground, for the tumour is smooth, freely movable and rarely more than 3 inches in diameter. If the tumour is not causing severe symptoms, the case can be watched and laparotomy is not called for. It is rare for such cysts to develop complications. It should be remembered that the removal of a corpus luteum cyst is not necessarily followed by abortion, presumably

because the placenta secretes progesterone almost at the beginning of pregnancy. Nevertheless there is always the risk of abortion if an ovarian tumour is removed during pregnancy. Ovarian cysts which cause an obstruction during the second stage of labour are dealt with by obstetricians.

[References to other titles are given under Ovary in the Index Volume. The subject is also dealt with under the heading of Ovary Diseases in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 318.]

OXYGEN THERAPY

BY RONALD V. CHRISTIE, D.Sc., M.D., F.R.C.P.
PROFESSOR OF MEDICINE, UNIVERSITY OF LONDON; PHYSICIAN,
ST. BARTHOLOMEW'S HOSPITAL, LONDON

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1. OXYGEN THERAPY IN PRESENCE OF CYANOSIS

255.] In general, the onset of cyanosis is the most important indication for oxygen therapy, but while the relief of cyanosis by oxygen may be spectacular in some cases, it is, unfortunately, imperceptible in others. It is important that the clinician should be able to recognize those patients who are likely to benefit from oxygen and those who are not, but this will be possible only if he appreciates the symptoms of anoxaemia and its dangers, and understands certain simple principles upon which oxygen therapy is based.

(1) First principle

The first principle concerns the dangers of oxygen lack. The more harmful anoxaemia is shown to be, the more important it is that oxygen should be given early and efficiently. Anoxaemia, if very severe, may result in permanent damage to the nervous system but, except in carbon-monoxide poisoning, this is rarely seen clinically. Much more common and more important is the effect of oxygen lack upon the heart. There is ample evidence that the degree of anoxaemia often seen in broncho-pneumonia, or acute heart failure, is sufficient to embarrass the heart of a healthy individual, and in these conditions relief of anoxaemia by means of oxygen may be a life-saving procedure. Since the cardiovascular system is the first to suffer, any assessment of the effects of oxygen therapy should be based partly upon careful observation of the pulse: a reduction in the rate of the heart beat or an improvement in the quality of the pulse is an important indication of successful treatment.

(2) Second principle

The second principle is that haemoglobin is almost completely saturated with oxygen when exposed to ordinary inspired air, and therefore the administration of oxygen to a healthy person does little to increase the quantity of oxygen taken up by the blood. In an individual whose arterial

blood is fully oxygenated in the lungs but whose hands are blue because they are cold, or who has widespread cyanosis due to a sluggish peripheral circulation, the administration of oxygen cannot be expected to relieve the cyanosis. Again, when the blood passing through certain parts of the lung is not aerated, the addition of oxygen to the air in the healthy parts of the lung will have but little compensatory effect. For instance, in a case of lobar pneumonia in which the left lung is consolidated and the right unaffected, any cyanosis that is present will be due to the flow of blood through the consolidated and unaerated left lung. If oxygen is given it can reach the blood passing through the solid left lung only by the very slow process of diffusion, and as the blood flowing through the healthy right lung is already fully aerated, no amount of oxygen will supersaturate it. On theoretical grounds therefore little relief of cyanosis can be expected from oxygen therapy when the anoxaemia is due to the flow of blood through a consolidated lung, and the same is true of other conditions, such as pneumothorax or massive collapse of the lungs, in which ventilation in parts of the lung is completely obliterated. Fortunately anoxaemia is seldom severe in these cases, since in the airless parts of the lung the pulmonary circulation rapidly diminishes and in time is almost obliterated. The latter phenomenon is well illustrated in the case of artificial pneumothorax, in which, even with extensive collapse, cyanosis is seldom seen.

Effect of areas of consolidation

In broncho-pneumonia or acute pulmonary oedema the situation is entirely different. Here the patches of consolidation are small or minute, and it is a deficiency of ventilation which causes anoxaemia. The quantity of air which enters the affected areas is insufficient to oxygenate the blood passing through them and this deficiency can be overcome effectively by enriching the inspired air with oxygen. The same is true of other conditions, such as asthma or emphysema, in which cyanosis is due to a generalized under-ventilation of the blood in the lungs. Oxygen, if properly given, should compensate for under-ventilation and should efficiently relieve anoxaemia.

Deficiency of ventilation

These generalizations can be re-stated in terms of physical signs; when moist sounds are plentiful and widespread, anoxaemia is often severe and oxygen, if properly given, should relieve cyanosis, but when added sounds are few or absent and bronchial breathing or suppression of breath sounds is conspicuous, oxygen is usually of little benefit.

Physical signs

(3) Third principle

The third principle is that lack of oxygen rarely, if ever, is the direct cause of dyspnoea. While the majority of patients who are ill and who complain of dyspnoea are also cyanosed, this by no means proves that the one is the cause of the other. Cyanosis is frequently said to be the cause of dyspnoea, not because of any proved relationship, but because it seems reasonable to suppose that the impaired respiratory function should be responsible for the respiratory symptoms. Nevertheless there is no foundation for such a supposition. In uncomplicated anoxaemia, such as is seen in carbon-monoxide poisoning or when flying at very high altitudes, dyspnoea comparable to that in pneumonia or cardiac failure is never observed, although the pilot may be incapacitated by oxygen want. Anoxaemia by its action upon the carotid sinus or gland may cause slight hyper-ventilation but not respiratory distress.

Relationship between cyanosis and dyspnoea

The practical bearing of these observations is that in pneumonia, and in other conditions in which dyspnoea is primarily due to an inflammatory lesion in the lung and not to cardiac failure, the relief of anoxaemia cannot be expected to produce any significant reduction in respiratory rate. For this reason the use of the symptom dyspnoea as an indication for oxygen therapy, or as a criterion of the efficacy of oxygen, is fallacious in most cases.

(4) Fourth principle

*Continuous
administration*

The fourth principle is that if oxygen is to be given at all, it should be given more or less continuously. The administration of oxygen for 10 minutes in every 2 hours to a patient who requires oxygen can only mean that he is denied the relief of anoxaemia for nine-tenths of the time.

These are the general principles which decide the therapeutic indications for oxygen therapy, and their application to disease is not difficult.

In severe broncho-pneumonia, acute pulmonary oedema and poisoning with the lung-irritant gases, there is exudate scattered throughout the lungs, and anoxaemia, which is frequently severe, can be relieved by oxygen. In this type of case, the early and efficient administration of oxygen may be a life-saving procedure.

*Value
questioned in
lobar
pneumonia*

In lobar pneumonia the therapeutic value of oxygen is in considerable dispute. There is no good evidence that oxygen therapy lowers the mortality rate or shortens the course of the disease, and even the relief of cyanosis is often imperceptible. When cyanosis is due to widespread consolidation little relief from oxygen is to be expected, but in the majority of patients with lobar pneumonia there are some areas in which consolidation is incomplete, and in these areas the administration of oxygen may be of some value. In other words, it is often impossible to state with certainty that oxygen will be of no value in a case of lobar pneumonia. It is usually better therefore to give oxygen a trial if the patient is cyanosed; if the patient dislikes this procedure or if it prevents sleep, it should be discontinued for a while and given again only if cyanosis increases. Fortunately, anoxaemia is seldom severe in lobar pneumonia.

After a large pneumothorax, hydrothorax or haemothorax, cyanosis is usually transient. If it persists, removal of air or fluid from the pleural cavity is indicated, rather than the administration of oxygen.

*Valueless in
long-standing
cyanosis*

Oxygen is of little or no value in the treatment of long-standing cyanosis such as is seen in chronic heart failure or emphysema, and should be reserved for the acute emergencies that frequently occur.

(5) Methods of administration

*Masks and
nasal tubes*

A concentration of 40 or 50 per cent of oxygen in the inspired air is usually considered adequate, and this can be achieved by several types of equipment now available. These can be divided into two groups—masks and tubes in the nose. Masks have the advantage of conserving the oxygen which is delivered during expiration and a sufficient concentration of oxygen can be obtained by a flow of from 3 to 4 litres a minute. Tubes, either as nasal catheters or carried on a spectacle frame, are often more convenient, but a flow of 6 or more litres of oxygen a minute is required, and this may produce dryness or irritation in the nasopharynx.

Oxygen tents are now seldom used, except in the case of children who will tolerate neither the mask nor nasal tubes.

2. OXYGEN THERAPY IN ABSENCE OF CYANOSIS

While the onset of cyanosis is the most important indication for oxygen therapy, there are three groups of clinical conditions without cyanosis in which the administration of oxygen is claimed to be beneficial.

(1) In anaemia

The first group concerns cyanosis masked by anaemia. The greater the degree of anaemia the greater may be the anoxaemia without cyanosis, until, when the haemoglobin falls to 30 per cent, the patient cannot be cyanosed although he may be suffering from a very dangerous degree of oxygen lack which can be relieved, partly at least, by oxygen. This clinical picture of anaemia and anoxaemia without cyanosis is uncommon except after operations on the lungs or wounds of the chest, but in these cases the administration of oxygen may be of value although cyanosis is not demonstrable.

(2) In diminished blood supply to the tissue

The second group concerns a recent innovation in oxygen therapy. When lack of oxygen is due solely to a diminished blood supply to the tissue, it had been assumed, until recently, that the administration of oxygen could be of little or no benefit. It seemed unreasonable that oxygen could in any way relieve anoxaemia when the blood passing through the lungs was already fully oxygenated, and the lack of oxygen in the tissues was due solely to a sluggish peripheral circulation. It is true that in this type of case oxygen therapy will not increase significantly the load carried by the haemoglobin, but if the oxygen is given in high concentrations the amount carried in the plasma may become significant. It has been claimed, and evidence is accumulating to support the claim, that in conditions such as shock when serious stagnant anoxaemia exists, the administration of pure oxygen may lead to demonstrable clinical improvement. This improvement is due solely to the increased load of oxygen carried in the plasma, and will occur, therefore, only when high concentrations of oxygen are administered. The use of ordinary equipment delivering between 40 and 50 per cent of oxygen cannot be expected to yield significant results; an efficient mask delivering almost pure oxygen must be used.

*Plasma
oxygen
content
increased*

(3) In air embolism, surgical emphysema and abdominal distension

The third and last group concerns another recent innovation in oxygen therapy. It is claimed that the inhalation of pure oxygen will accelerate the absorption of any collection of gas which is trapped in the body. The administration of oxygen in high concentrations is therefore advised in conditions such as air embolism, surgical emphysema and severe abdominal distension. The use of oxygen for this purpose is based upon sound physiological principles. Under normal conditions about 80 per cent of the gas pressure in the tissues is exerted by nitrogen. If most of the nitrogen in the plasma is replaced by oxygen, as occurs when pure oxygen is breathed, a precipitous fall

*Acceleration
of gas-
absorption
rate*

*Fall in gas
pressure*

in gas pressure occurs where the plasma reaches the tissues and the oxygen is used. The total pressure of gas in the tissues is then reduced to a level very much below that of the atmosphere, with the result that the rate of absorption of any air trapped in the body will be greatly accelerated.

[References to other titles are given under Oxygen Therapy in the Index Volume.]

PAIN—CAUSALGIA

By A. M. BOYD, F.R.C.S.

PROFESSOR OF SURGERY, UNIVERSITY OF MANCHESTER; SURGEON AND DIRECTOR
OF SURGICAL CLINICAL UNIT, ROYAL INFIRMARY, MANCHESTER

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1. DEFINITION

256.] The term causalgia should be restricted to a syndrome appearing after injury to a peripheral nerve containing sensory fibres, characterized by burning pain referred to the peripheral sensory distribution of the injured nerve and completely relieved by sympathetic denervation of the limb. An essential feature of the pain is its aggravation by changes in the patient's environment.

2. GENERAL CONSIDERATIONS

Weir Mitchell introduced the term causalgia in 1864 to describe an intense burning pain which occasionally arose after injuries to peripheral nerves containing sensory fibres. His description of the syndrome was quite specific. The recent tendency to include other post-traumatic painful states has resulted in much confusion. The concentration of nerve injuries in special centres early in World War II permitted the extensive study of post-traumatic painful states. The various types of pain following peripheral nerve injuries, Sudeck's atrophy, painful osteoporosis, the "minor" causalgias, and the painful phantom limb are believed by many authors to be manifestations of the same underlying disorder. In the opinion of the writer, causalgia is a definite clinical entity which should be segregated from this miscellaneous group of painful post-traumatic dystrophies.

3. AETIOLOGY

Incidence Causalgia was observed in about 5 per cent of peripheral nerve injuries admitted to special centres. The true incidence, however, is probably considerably less. By no means all the cases of nerve injuries reach special centres. The term causalgia, moreover, has been applied rather loosely to other painful sequelae of nerve injuries. It has been commonly stated that causalgia occurs only in anatomically incomplete lesions. The misconception is largely due to the fact that incomplete lesions are much more common than complete ones. Causalgia can occur in anatomically complete lesions, and rarely, even, in a phantom limb.

Nerves involved In an overwhelming majority of cases the nerves involved are the tibial component of the sciatic and the median. Rarely, causalgia has been reported following lesions of the peroneal, saphenous, radial and ulnar nerves. With the possible exceptions of those following ulnar nerve injuries, the painful states after injury to the other nerves do not appear to conform with the rigid definition advocated by the writer.

Type of injury Causalgia is very rare after closed nerve injuries. The syndrome is almost confined to penetrating wounds, usually caused by high-velocity missiles. The view that mild infection of the wound is an important factor does not accord with the fact that in many cases causalgia is of instantaneous onset. In a few cases coincidental injuries to major blood-vessels are found.

Location of wound The site of the nerve injury is, however, significant. In true causalgia the wound is most commonly in the proximal half of the limb: in the lower extremity, in the buttock and thigh; in the upper extremity, in the axilla, upper arm and elbow region. Painful states following injuries to nerves in the distal part of the extremities show characteristic features which differentiate them from true causalgia.

Pathogenesis of causalgia The various views may be considered under three headings, according to the source of the pain.

(1) In the tissues of the area in which the pain is referred

Attention is naturally focused on the area in which the pain is felt and in which the striking vascular and atrophic changes are observed.

(a) *Ascending neuritis*

Weir Mitchell (1874) attributed the symptoms to an ascending neuritis.

(b) *Vascular stasis*

(i) *Vasoconstriction*.—Leriche (1939) believed that the irritation of the axons by scar tissue in the damaged nerve acted as a continuous stimulus to the vasomotor nerves, resulting in vasoconstriction both in the nerve itself and in the area of its distribution.

(ii) *Vasodilatation*.—Lewis (1936) believed that chronic irritation of the nerve by the scar tissue stimulated the sensory nerve fibres to antidromic action, resulting in the liberation of substances in the skin which induce a state of local erythralgia, and that it is from the skin that the pain arises.

(2) In the central nervous system

It is thought by some authorities that the barrage of sensory impulses leads to changes in the activities of the regulatory centres of the central

nervous system, which enable them eventually to maintain the sensation of pain.

(3) In the nerve lesion

Artificial synapse

The possibility of conduction across an artificial synapse has been proved (Granit, Leksell and Skogland, 1944). Doupe, Cullen and Chance (1944) suggest that the pain is caused by efferent impulses in the sympathetic fibres stimulating the afferent sensory fibres at the site of the nerve lesion.

(4) Discussion

Any theory of the genesis of causalgia must explain: (1) its occurrence in a complete nerve lesion in which there is no sensory pathway between the painful area and the brain, (2) its occurrence in a phantom limb, and (3) its relief by sympathectomy.

Consideration of these facts proves that the origin of the pain must be in the nerve lesion. Moreover, causalgia is completely relieved by excision of the injured segment of nerve.

4. CLINICAL FEATURES

The outstanding feature is the intense burning pain referred to the whole or part of the peripheral cutaneous distribution of the injured nerve. Other clinical features often ascribed to causalgia—trophic changes and the vascular state of the part—are entirely dependent upon the nerve lesion and are seen in nerve injuries uncomplicated by causalgia.

Pain may begin immediately on wounding, or the onset may be delayed for a few hours or a few weeks. Kirklin, Chenoweth and Murphy (1947) report instantaneous onset in 80 per cent of their cases, Mayfield (1947) in 50 per cent of his cases. In a personal series of 10 cases instantaneous onset was noted in 2 patients. If the patient is temporarily dulled by the wound or by heavy sedation, pain may not be noticed for some hours. In the remaining cases pain begins within the first 4 weeks. Late onset of pain after many weeks or months is more likely to be due to the other painful post-traumatic states than to true causalgia.

The pain is burning in character, variously described by patients as resembling searing with a red-hot iron or stabbing with red-hot needles. In the upper extremity the pain is confined roughly to the area of sensory distribution of the median nerve in the hand. It is always most severe in the centre of the palm just proximal to the heads of the second and third metacarpal bones, radiating to a varying extent into the outer $3\frac{1}{2}$ digits. In the lower limb the pain is worse in the ball of the foot, radiating into the inner 3 toes. It is always severe and often agonizing, but in milder cases is bearable.

The severity varies, but it is usually as severe at its onset as it ever becomes. Although the pain is continuous in the sense that it is never entirely absent, various factors aggravate it and may lead to paroxysms of intense agony.

The aggravation of the pain by factors in the patient's environment is an essential feature of true causalgia and is, therefore, included in the definition of the syndrome. The aggravating factors are of two types.

(1) Those commonly found in all cases of causalgia—fright, sudden movements, jarring or touching the affected part.

(2) Factors specific to the individual—certain noises, such as the banging of doors, the rustling of paper, or even the sounds of certain words, also light and various emotional stimuli.

Relief of pain

Pain is relieved if the part is kept absolutely still and protected from external stimuli. The patient, therefore, isolates himself in a quiet corner of the ward, protected so far as is possible from draughts, noises, bright lights and so on, grasping the affected limb at the wrist or ankle in order to keep it still. Most patients with causalgia find relief from cooling the part; therefore they wrap the limb in a cloth soaked in cold water. Some patients, however, claim alleviation from pain by warmth. Whether they get relief from heat or cold depends entirely upon the vascular state of the part. Most commonly the skin is hot, red and hyperalgesic—a state of erythralgia, relief from which is obtained by cold. Occasionally the area to which the pain is referred is cold and cyanotic, in which case warmth diminishes the discomfort.

Effect of temperature

Effect of posture

As a rule, elevation eases the pain and dependency intensifies it. Again, the effect of posture is dependent upon the local vascular conditions. When there is erythralgia the patient elevates the limb, but in the rarer cases in which the painful area is cold and cyanotic, dependency gives relief.

Other clinical features

The cutaneous area to which the pain is referred is usually hyperaesthetic. The skin is hot, red, shiny and hairless, and when warm is drenched in perspiration. On cooling, the skin is mottled red and blue, and is covered with minute droplets of sweat. There is always wasting of the tissues, particularly of the subcutaneous fat, best seen in the fingers where the nails (which the patient dare not cut) curve over the wasted finger pads. The digits are held slightly flexed and rapidly become stiff. There is marked osteoporosis of the phalanges. The part may, however, be hypo-aesthetic or anaesthetic, dry, cold and cyanotic. Nathan (1947) stresses the fact that these changes are entirely dependent upon the peripheral nerve injury and are in no way specific to causalgia. The nerve lesion is most commonly a partial, irritative lesion: hence the common appearance of hyperaesthesia, vasodilatation, sweating and trophic changes. When, however, the nerve lesion is complete, these clinical features, so often considered characteristic of causalgia, are not seen.

Mental state

An account of the clinical features of causalgia cannot be concluded without some reference to the mental state of the sufferer. The view that causalgia occurs only in the highly-strung, tense, emotional individual dies hard. There is still the feeling that there is something vaguely reprehensible, almost amounting to malingering, about the causalgic patient, which tends to alienate the sympathy of medical attendants and nursing staff. The prolonged agony, lack of sleep and malnutrition make the sufferer introspective, suspicious, refractory and extremely difficult to manage. The complete and dramatic return to normality after relief of pain, however, reveals the essentially or-

Central
ination
after cure by sympathectomy has failed to reveal any conscious emotional make-up, although it may be conceded that in the highly-strung emotional individual the exacerbations may be worse than in the more stolid type.

5. DIAGNOSIS

The diagnosis of causalgia is extremely simple and depends upon the following factors:

- (1) The appropriate peripheral nerve lesion.
- (2) The characteristic burning pain, the essential features being the paroxysmal character and the aggravation caused by disturbing the patient's environment.
- (3) The relief which commonly follows blocking the sympathetic trunks with procaine and lasts for the duration of the sympathetic paralysis or longer.

If the above criteria are present the diagnosis of causalgia is established.

6. DIFFERENTIAL DIAGNOSIS

Attention has already been drawn to the confusion which has resulted from the inclusion of other painful post-traumatic syndromes under the term causalgia. From the maelstrom of post-traumatic painful states can be separated those which follow lesions of the peripheral nerves. Four groups may be recognized.

(1) Causalgia—a specific syndrome, the diagnostic criteria of which can be *Causalgia* recognized as established.

(2) Tactile hyperpathia, in which the pain is of the "pins and needles" type. *Tactile hyperpathia* It is felt only when the affected area is touched. Tactile hyperpathia may follow any lesion of a peripheral nerve containing sensory fibres. It is most commonly seen after injuries to the digital nerves.

The sufferer may be entirely incapacitated. Prolonged voluntary immobilization leads to nutritional changes in the tissues and stiffness of joints. The pain is uninfluenced by Novocain block of the sympathetic trunk.

(3) Deep cold pain. Kellgren (1947) in an extensive study of post-traumatic *Deep cold pain* pain has isolated a group in which the pain is aching in character, accompanied by deep hyperalgesia. The pain is made worse by cold and relieved by heat.

Deep cold pain is often greatly improved by sympathectomy. Kellgren (1947) suggests that sympathectomy, by abolishing reflex vasoconstriction, interferes with the normal cooling of the extremity and thus relieves spontaneous pain, the deep hyperalgesia remaining unaltered. The importance of "cold" pain in the differential diagnosis of causalgia lies in the fact that there is a tendency to include under the term "causalgia" all post-traumatic pain improved by sympathectomy. Cold pain may follow injuries apart from those involving peripheral nerves. The pain is an intense ache increased by cold and relieved by warming and by sympathectomy. It is deep in quality and diffuse in distribution, with a tendency to proximal reference. There is abnormal sensitivity of the deep tissues at the source of the pain to mechanical stimuli.

(4) Phantom pain is of central origin and is associated with the patient's *Phantom pain* plastic image of the affected part. The reflex exacerbations typical of causalgia are absent and interruption of the sympathetic nervous supply leaves the pain unaltered.

7. TREATMENT

*Early
treatment
essential*

Spontaneous disappearance of the pain in causalgia has been described, but treatment should never be delayed in anticipation of this occurrence. Immediate treatment as soon as the diagnosis is established is imperative in order to forestall the mental and physical deterioration consequent upon prolonged suffering, and to prevent the stiffness, loss of function and nutritional changes following prolonged immobilization. Complete sympathetic denervation of the affected extremity gives immediate, complete and permanent relief.

(1) Conservative treatment

*Active
exercises*

It is considered by some authorities that the vascular and atrophic changes are the cause rather than the consequence of the pain. They advocate active exercises in the early stages, undertaken with the affected extremity immersed in warm water after procaine block of the painful area, affected nerve or sympathetic trunks, combined with suitable psychotherapy (Livingstone, 1943). Conservative treatment of this type is extremely useful in many of the ill-defined painful post-traumatic dystrophies and so-called "minor" causalgias, but has no place whatsoever in the management of true causalgia.

(2) Operative treatment

(a) Operations on the nerve lesion

Resection of the injured segment of nerve, with suture, will give complete relief, but requires prolonged after-treatment and involves great delay in restoration of function, with a greater or lesser degree of permanent disability. External and internal neurolysis rarely relieves the pain, and also requires the active co-operation of the patient in the all-important rehabilitation which is impossible if pain is unrelieved. Local operation on the nerve lesion should be undertaken only if the nerve injury, on its own merits, requires it, and then only after pain has been abolished by sympathectomy in order to secure the patient's full co-operation in restoration of function.

(b) Periarterial neurectomy

There have been a number of successful results reported after periarterial neurectomy, originally advocated by Leriche (1939). Gask and Ross (1937), in their monograph, report success in 3 out of 5 cases. In the remaining 2 patients pain recurred after a few weeks but was completely abolished by ganglionectomy. Periarterial neurectomy is an incomplete form of sympathectomy and is no longer practised.

(c) Procaine block of the sympathetic trunk

Occasionally procaine block of the sympathetic trunk, undertaken to establish the diagnosis, has resulted in permanent relief of the symptoms.

(i) *Procaine sympathetic block of the upper extremity: technique.*—The patient sits in a chair with the head and neck flexed. The spinous processes of the seventh cervical and first thoracic vertebrae are identified (Figs. 232 and 233). An 8-centimetre Labat needle is inserted 4 centimetres lateral to the mid-point between the seventh cervical and first thoracic spines. The needle is passed medially at an angle of about 20 degrees until the first rib or first thoracic transverse process is felt, usually at a depth of about 3 or 4 centimetres.



FIG. 232.—Showing the relation of the spine of the seventh cervical vertebra to the transverse process of the first thoracic vertebra.

FIG. 233.—Showing point of injection for procaine sympathetic block of the upper extremity.

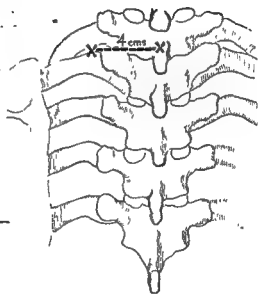
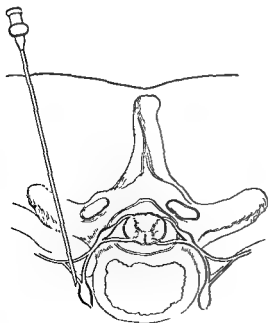


FIG. 234.—Procaine sympathetic block of the upper extremity.

The point of the needle is passed beneath the transverse process and injection begun in order to strip up the pleura. The side of the body of the first thoracic vertebra is felt after advancing the needle a further 3 centimetres or so, and 10 cubic centimetres of 2 per cent procaine are injected (Fig. 234).

(ii) *Procaine block of the lumbar ganglia: technique.*—The patient lies on his side with a pillow under his loin. An intradermal weal of 2 per cent procaine



FIG 235 —Showing point of injection for procaine block of the lumbar ganglia.

is raised at a point opposite the second lumbar spine near the outer border of the erector spinae muscle (Fig. 235). A 12-centimetre Labat needle is inserted at an angle of 30 degrees from the horizontal plane. By this technique the

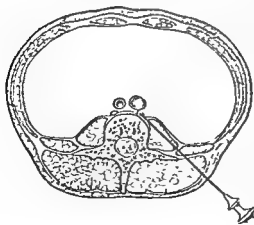


FIG 236 —Procaine block of the lumbar ganglia.

transverse processes are usually avoided. The needle is advanced through the erector spinae and psoas muscles until the anterolateral aspect of the body of the second lumbar vertebra is felt, usually 9 or 10 centimetres from the surface, and 20 cubic centimetres of 2 per cent procaine are injected (Fig. 236). The procaine flows up and down the gutter formed by the aorta or vena cava, peritoneum, and psoas muscles. Only one puncture is required to produce a complete paravertebral block.

Shumacker, Speigel and Upjohn (1948) reported permanent relief in

21 out of 83 patients after one or more procaine blocks. They made the important observation that there was little likelihood of permanent cure

from repeated blocks if cessation of pain lasted only for the duration of the sympathetic paralysis, or if after successive blocks there were decreasing periods of relief.

If, however, after the first block, relief of pain lasted longer than the sympathetic paralysis, and if the period of relief lengthened with further blocks, permanent cure might result.

Repeated procaine blocks of the sympathetic trunk should be tried if the duration of relief from pain outlasts the sympathetic paralysis.

(d) Sympathectomy

Complete and permanent cure follows thorough interruption of the sympathetic outflow to the affected extremity. Incomplete relief of pain, or occasionally complete failure to abolish the symptoms, has been shown to be due to inadequate sympathetic denervation. It must be remembered that it is necessary to denervate the *site of the wound in the nerve* and not simply the area to which the pain is referred.

All cases of causalgia in which the diagnosis has been established by procaine block of the sympathetic trunk are suitable for sympathectomy. Indications

(3) Operative technique

A radical sympathetic denervation of the upper extremity may be carried out by a posterior or an anterior approach. The preganglionic sympathectomy described by Smithwick (1936) is usually considered to be the most radical. The writer's preference, however, is for the anterior operation (Telford, 1935), in which the sympathetic trunk below the third thoracic ganglion and the rami to the third and second ganglia are divided. There is less post-operative pain and shorter hospitalization with the anterior approach. (See Autonomic Nervous System, Vol. 1, p. 472.) Upper extremity

The first, second and third lumbar ganglia should be removed. It has been shown by Jepson and Ratchiffe (1947) that the first lumbar ganglion must be included in order to be certain of complete sympathetic denervation of the limb. Lower extremity

8. RESULTS

The results of sympathectomy in true causalgia are excellent. Incomplete relief of pain is most commonly due to inadequate denervation or to incorrect diagnosis. It is essential to distinguish true causalgia from other causes of post-traumatic pain.

REFERENCES

- Doupe, J., Cullen, C. H., and Chance, G. Q. (1944). *J. Neurol. Neurosurg. Psychiat.*, 7, 33.
- Gask, G. E., and Ross, J. P. (1937). *Surgery of the Sympathetic Nervous System*, 2nd ed. London; Baillière, Tindall & Cox.
- Granit, R., Leksell, L., and Skogland, C. R. (1944). *Brain*, 67, 125.
- Jepson, R. P., and Ratchiffe, A. H. (1947). Personal communication.
- Kellgren, J. H. (1947). *Brain*, 70, 100.
- Kirklin, J. (1947). *Surgery*, 21, 321.
- Lersch, J. (1947). *Surgery*, 21, 321.
- Young, Baltimore; Williams & Wilkins.
- Lewis, T. (1936). *Peripheral Vascular Disorders*. London; Macmillan.

- Livingstone, W. K. (1943). *Pain Mechanisms*. New York; Macmillan.
- Mayfield, F. H. (1947) *Ann. Surg.*, 74, 522.
- Mitchell, S. W. (1874). *Injuries of Nerves and their Consequences*. Philadelphia; Lippincott.
- Nathan, P. W. (1947). *Brain*, 70, 145.
- Shumacker, H. H., Spiegel, J. I., and Upjohn, R. H. (1948) *Surg. Gynec. Obstet.*, 86, 76.
- Smithwick, R. H. (1936). *Ann. Surg.*, 104, 339.
- Telford, E. D. (1935). *Brit. J. Surg.*, 23, 448.
- [References to other titles are given under Pain—Causalgia, in the Index Volume. The subject is also dealt with under the heading of Pain in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 359.]

PANCREAS

By C. F. W. ILLINGWORTH,

C.B.E., M.D., CH.M., F.R.C.S. Ed., F.R.F.P.S.

REGIUS PROFESSOR OF SURGERY, UNIVERSITY OF GLASGOW

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1. ACUTE PANCREATITIS : ACUTE PANCREATIC NECROSIS

257.] The term acute pancreatitis is now recognized to be a misnomer. The disease to which it is applied is not an inflammation but an acute necrosis due to activation of trypsinogen within the gland. It is thought that such activation is usually due to reflux of infected bile, an occurrence rendered possible in the majority of subjects by the anatomical disposition of the terminations of the biliary and pancreatic ducts. This view was first put forward by Opie in 1901 on the basis of a necropsy finding of a minute gall-stone impacted at the duodenal papilla, and although subsequent experience has shown that such a stone is rarely to be found, it is probable that the same effect may be produced by spasm of the sphincter of Oddi. In other cases it is thought that the tryptic ferment may be activated by blood-borne agents. The occasional development of pancreatitis as a complication of mumps supports this view.

- Livingstone, W. K. (1943). *Pain Mechanisms*. New York; Macmillan.
- Mayfield, F. H. (1947). *Ann. Surg.*, **74**, 522.
- Mitchell, S. W. (1874). *Injuries of Nerves and their Consequences*. Philadelphia; Lippincott.
- Nathan, P. W. (1947). *Brain*, **70**, 145.
- Shumacker, H. B., Spiegel, J. I., and Upjohn, R. H. (1948). *Surg. Gynec. Obstet.*, **86**, 76.
- Smithwick, R. H. (1936). *Ann. Surg.*, **104**, 339.
- Telford, E. D. (1935). *Brit. J. Surg.*, **23**, 448.

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By C. F. W. ILLINGWORTH,

C.B.E., M.D., CH.M., F.R.C.S. Ed., F.R.F.P.S.

REGIUS PROFESSOR OF SURGERY, UNIVERSITY OF GLASGOW

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1. ACUTE PANCREATITIS : ACUTE PANCREATIC NECROSIS

257.] The term acute pancreatitis is now recognized to be a misnomer. The disease to which it is applied is not an inflammation but an acute necrosis due to activation of trypsinogen within the gland. It is thought that such activation is usually due to reflux of infected bile, an occurrence rendered possible in the majority of subjects by the anatomical disposition of the terminations of the biliary and pancreatic ducts. This view was first put forward by Opie in 1901 on the basis of a necropsy finding of a minute gall-stone impacted at the duodenal papilla, and although subsequent experience has shown that such a stone is rarely to be found, it is probable that the same effect may be produced by spasm of the sphincter of Oddi. In other cases it is thought that the tryptic ferment may be activated by blood-borne agents. The occasional development of pancreatitis as a complication of mumps supports this view.

(1) Pathological features

In severe cases the pancreas is swollen, brawny and indurated. There may be extravasation of blood into the retroperitoneal tissues, owing to erosion of blood-vessels by escaped trypsin. The gall-bladder is tense with muddy bile, and often contains multiple small stones. The peritoneal cavity contains a turbid or blood-stained exudation. Yellow-white plaques of saponified fat abound in the omental and extraperitoneal tissues. In milder cases the changes are less marked, and in the mildest, induration of the pancreas with some oedema and a few scattered points of fat necrosis are the only features of note.

(2) Clinical features

In the most fulminating cases the disease starts with dramatic suddenness. Often it begins within half an hour of a heavy meal. The patient is suddenly seized by an intense lancinating pain in the upper abdomen, sometimes radiating through to the back. Persistent retching is a distinctive feature. Often there is nausea and there may be repeated vomiting of small quantities of bilious fluid. The toxic effects are severe and lead to marked collapse, often accompanied by lividity and cyanosis. Abdominal examination shows diffuse muscular rigidity and tenderness, most marked in the epigastrium.

In milder cases the pain is less agonizing and the toxic manifestations are less severe. The picture may resemble that of acute cholecystitis except that the pain tends to be more marked towards the left side and retching and hiccup are more pronounced.

(3) Differential diagnosis

Acute pancreatitis may closely resemble perforated peptic ulcer and, indeed, may be indistinguishable from this condition on clinical grounds. It is true that in pancreatitis the pulse rate tends to be more rapid at an early stage, that the rigidity is not so board-like as in perforation, and that lumbar pain is sometimes present, but none of these features is sufficiently characteristic to be relied upon. The radiological demonstration of free gas under the diaphragm will point to perforation, but its absence is not significant. If the diastase test (see below) can be carried out it may establish the diagnosis, but this usually involves a dangerous loss of time, and in practice if the distinction cannot be made otherwise there should be no hesitation in performing a laparotomy.

The possibility of coronary thrombosis may cause confusion in diagnosis if the pain is referred to the upper abdomen. Generally, however, radiation of the pain to the praecordia and left arm, the more profound collapse and the fall of blood-pressure serve to establish the diagnosis. Pneumonia and diaphragmatic pleurisy may be distinguished by the increased respiratory rate, the catch on respiration and the absence of true abdominal rigidity. In milder cases acute pancreatitis must be differentiated from acute cholecystitis, leaking peptic ulcer and subacute obstruction.

(a) The diastase test

The diastase test consists in the estimation of diastase or amylase in the urine. The standard method is to add 1 part of urine to 4 parts of a standard starch solution, and incubate. Samples are withdrawn at intervals and tested with

iodine. The diastase index is determined by a calculation based on the time required for the blue colour to disappear.

(b) *Loewe's mydriasis test*

Loewe's mydriasis test is now recognized to be unreliable and should not be employed.

(4) **Treatment**

It is now established that in its less severe forms acute pancreatitis will subside under conservative treatment, and many surgeons believe that even in the more severe forms the mortality is no higher under conservative than it is under operative treatment. It must be remembered, however, that this impression was gained before the value of massive infusions of saline, proteins, plasma and blood was established, and in those days the risk of operation in the presence of such a degree of shock as is seen in pancreatitis was formidable. It must be recognized also that in the more severe forms there is often some doubt as to the diagnosis. Finally, a not unimportant point, it is a common experience that the agonizing pain can be relieved only, and is relieved promptly, by laparotomy. For these reasons, in the writer's opinion, the undoubted risks should not be allowed to weigh too heavily against operation.

Conservative and operative treatment assessed

When the abdomen is opened the diagnosis is established readily by demonstrating areas of fat necrosis. It is sometimes advised to incise the peritoneum over the pancreas and insert a drain, but the value of this step is doubtful. In view of the mode of origin of the disease it seems more rational to drain the distended gall-bladder in order to relieve tension within the biliary passages. The irritant peritoneal exudate should be evacuated.

In less fulminating cases in which operation is demanded for the continuing pain, the gall-bladder and common bile-duct should be explored for stone. There is some reason to believe that the liability of pancreatitis to recur may be diminished by performing cholecyst-duodenostomy or cholecyst-jejunostomy.

2. PANCREATIC CYST

The only type of cyst requiring consideration here is the "false" cyst. This is a "False" collection of fluid in the lesser peritoneal sac (omental bursa), which is generally ascribed to injury to the pancreas or to a mild localized pancreatitis. There may be a history of a blow on the abdomen, or of an acute attack with epigastric pain and vomiting. Thereafter the cyst gradually enlarges and may attain a great size. Generally it projects forwards, displacing the stomach upwards and the transverse colon downwards, or it may come towards the surface above the stomach or below the colon. The symptoms include anorexia, epigastric discomfort, occasional vomiting, some loss of weight and impairment of the general health. The swelling is readily palpable in the epigastrium, generally to the left of the midline. The diagnosis is indicated by its cystic character and its relation to the stomach and colon, as determined clinically or radiographically.

Symptoms

A small cyst may sometimes be palpable in the epigastrium.

its wall to the anterior parietal peritoneum and establish drainage. In order to establish dependent drainage it is generally better, after evacuating the cyst, to insert a tube through a stab wound in the left loin. Since the fluid may contain trypsin and may continue to discharge for a considerable time, care must be taken to prevent digestion of the skin adjacent to the site of drainage. If such digestion occurs the treatment is as for pancreatic fistula (see p. 443).

3. CARCINOMA OF THE PANCREAS

Carcinoma of the pancreas occurs mainly in men and usually after the age of 50 years, though exceptionally as early as the thirtieth year.

(1) Point of origin

(a) *The body or tail of the pancreas*

When a carcinoma arises in the body or tail of the pancreas the early symptoms are obscure, including vague indigestion, anorexia and loss of weight. Later, pain develops and may be severe. Owing to the deep-seated situation the growth is usually not palpable until a later stage, and often the first sign on abdominal examination is enlargement of the liver which results from metastases. X-ray examination may show displacement or invasion of the stomach or the terminal part of the duodenum. Generally there is no treatment. Exceptionally in an early case the body and tail of the pancreas may be removed.

*Enlargement
of liver*

(b) *The head of the pancreas*

Carcinoma of the head of the pancreas is more common and more readily diagnosed. The outstanding feature is jaundice, and this may be the first to develop, though more often there is a history of some lack of energy, with anorexia and slight loss of weight for a few weeks previously. In about 50 per cent of cases there is some pain, usually a dull ache or a feeling of heaviness or distension in the epigastrium.

Jaundice

Nearly always the jaundice is insidious in onset, deepens progressively and ultimately assumes a dark olive-green hue. Exceptionally it may fluctuate a little, or even disappear for a time. The usual concomitant symptoms of obstructive jaundice are present—pigmentation of the urine, pallor of the stools and pruritus—but the temperature is normal. Owing to its deep situation the tumour is rarely palpable. Usually the liver is enlarged as a secondary result of the biliary obstruction, and its margin may be palpated an inch or so below the costal margin. Distension of the gall-bladder is usual, following Courvoisier's law, but if the gall-bladder is deeply placed under the liver it may not always be palpable.

(c) *Peri-ampullary carcinoma*

In peri-ampullary carcinoma—a term applied to growths arising from the ampulla of Vater, the duodenal papilla or the termination of the common bile-duct, as well as to growths of that part of the pancreas immediately related to the ampulla—the symptoms are usually similar to those previously enumerated, but certain additional features may be present. These are: (i) fluctuation in the jaundice due to superficial necrosis of the growth; (ii) infection of the ulcerated surface of the growth leading to cholangitis with recurring fever, in contrast with the afebrile course characteristic of the typical

*Additional
symptoms*

pancreatic carcinoma; (iii) occult blood in the stools; and (iv) a filling defect in the duodenum, revealed on x-ray examination after a barium meal.

(2) Indications for operation

Although the results of surgical treatment are, on the whole, disappointing, there are good reasons why operation should always be advised, provided, of course, that the patient's general condition is satisfactory and there is no evidence of secondary growths. The diagnosis is not always beyond doubt, and occasionally exploration may reveal such lesions as chronic pancreatitis or a stone in the common duct; further, if the diagnosis is confirmed, it is often possible to perform a palliative anastomosis, and in rare cases radical extirpation is feasible.

(3) Pre-operative preparation

The two main dangers in operation on the jaundiced patient are those from hepatic failure and from haemorrhage. No specific treatment is available to mitigate the effects of liver damage, but benefit is gained by the administration of sugar to replenish the glycogen reserve, which is greatly diminished in all liver disease. If the patient can take food the sugar may be given in the form of sucrose in orange or lemon drinks, tea or other beverages. If not, glucose must be given intravenously. Should glycosuria result, the glucose must be backed by appropriate doses of insulin.

*Risk of
hepatic failure
and
haemorrhage*

Vitamin K is highly effective in preventing haemorrhage in all except the most severe cases, and since it is entirely free from danger it should be given as a routine in all cases. The dose is 10 milligrams intramuscularly, twice or thrice daily, beginning a day or two before operation and continuing for a week thereafter, or longer if the jaundice persists unrelieved. Prothrombin tests are unnecessary and may be misleading, as the prothrombin index may be normal before operation and fall very rapidly thereafter.

*Prothrombin
tests
inconclusive*

(4) Exploration

A right paramedian incision is usually adequate; a Kocher incision below and parallel to the right costal margin is used if wider access is desired. Attention should be directed first to the gall-bladder; if the diagnosis has been correct it will nearly always be distended, thin-walled and dark due to the deeply pigmented bile within it. The supraduodenal part of the common bile-duct will also be distended, thin-walled and of a deep green colour.

Access to the pancreas is gained by tearing through the thin lesser omentum, or by making an opening in the gastro-colic omentum. Often it is advisable also to mobilize the duodenum by dividing the peritoneal reflection on its outer side and sponging it forwards and medially. When this has been done the head of the pancreas can be palpated between the fingers and thumb.

Experience shows that the diagnosis of pancreatic lesions is by no means easy, and there are many cases on record in which the finding at operation of an apparently malignant tumour was belied by a long post-operative survival. In such circumstances it is generally assumed that the actual disease has been chronic pancreatitis, but doubtless cysts and chronic abscesses of the pancreas and induration round a stone impacted in the common duct may sometimes have been responsible. In view of this experience it is clearly desirable to obtain microscopic confirmation. A biopsy, therefore, should be

*Need for
microscopic
confirmation*

performed, a small wedge of tissue being removed from the affected part. Haemorrhage is often troublesome, but with care it can be controlled by inserting 2 or 3 fine catgut sutures.

(5) Palliative operation

Anastomosis

In most cases the best that can be done is to relieve the jaundice by anastomosing the gall-bladder to some part of the alimentary tract. Although, if carcinoma is present, such a procedure does not greatly prolong life it has valuable ameliorative effects, for it improves the well-being of the patient, relieves the mental depression which jaundice induces and, moreover, gives immediate relief to the pruritus. Its value is, of course, obvious when, despite the appearance at operation, the condition actually proves to be non-malignant.

The procedure most often followed is to anastomose the gall-bladder to the stomach. This has been criticized on the grounds that in some cases it may lead to an ascending infection of the biliary passages, and that in other cases the stoma contracts and is eventually obliterated. Similar disadvantages attach to cholecyst-duodenostomy. It has been claimed that cholecyst-jejunostomy avoids these complications, particularly if it is performed in such a way as to interpose a segment of empty jejunum between the gall-bladder and the food passages. On the other hand, however, the disadvantages of cholecyst-gastrostomy count for little in the comparatively brief survival period of patients with malignant disease, and since this operation is the most simple to perform and carries the lowest immediate mortality, it may well be recommended for general use.

Emptying of gall-bladder

Whichever procedure is chosen, as a first step the distended gall-bladder must be emptied of its thick viscous biliary content. A wide-bore trocar and cannula should be used. It is introduced on the inferior aspect of the gall-bladder about an inch proximal to the fundus so that the puncture can be used conveniently for the subsequent anastomosis. Evacuation of the bile is facilitated by connecting the cannula with a suction apparatus.

(a) Cholecyst-gastrostomy

In cholecyst-gastrostomy (Fig. 237) the gall-bladder, now completely empty and relaxed, is apposed to the anterior wall of the stomach at a convenient

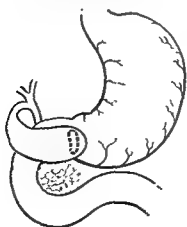


FIG. 237.—Cholecyst-gastrostomy.

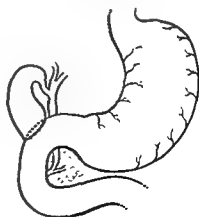


FIG. 238.—Cholecyst-duodenostomy.

point in the pyloric antrum. The anastomosis is made by the usual four-layer *Four-layer technique* technique. The stoma should measure at least 1 inch in diameter. Great care must be taken in applying the outer rows of sutures to avoid puncturing the thin-walled gall-bladder. The sutures must be applied closely and evenly in order to bring the two serous surfaces into wide and intimate contact, so as to ensure a watertight anastomosis.

(b) *Cholecyst-duodenostomy*

In cholecyst-duodenostomy (Fig. 238) the procedure is similar. It may be necessary to mobilize the duodenum in order to bring the gall-bladder into apposition without tension.

(c) *Cholecyst-jejunostomy*

In cholecyst-jejunostomy (Fig. 239) the gall-bladder may be anastomosed end to side to a loop of jejunum brought up in front of the colon. If so the two limbs of jejunum at the base of the loop should be joined by side-to-side anastomosis. A preferable alternative is to divide the jejunum about 8 inches from the duodeno-jejunal flexure, insert the upper cut end into the jejunum some 8 inches lower, and anastomose the lower cut end to the fundus of the gall-bladder. By this method the biliary tract is separated

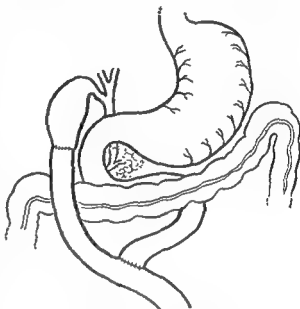


FIG. 239.—Cholecyst-jejunostomy.

from the food passages by an 8-inch segment of jejunum and the risk of infection is thus minimized. *Risk of infection minimized*

(6) Radical resection of the head of the pancreas

Radical resection (Fig. 240) is practicable in carcinoma arising in the region of the ampulla, or in the head of the pancreas provided that it has not extended beyond the pancreas and that metastases are not present.

The operation may be carried out in one or two stages. The one-stage method carries certain advantages and may be advised if the patient's general condition is good and the jaundice of short duration. In other cases the two-stage procedure is preferable.

In the two-stage procedure, at the first session the biliary obstruction is short-circuited, preferably by anastomosing the gall-bladder to the jejunum, and if thought desirable a gastro-jejunostomy may also be performed in preparation for the second stage. The technique of the second stage is similar to that of the one-stage procedure.

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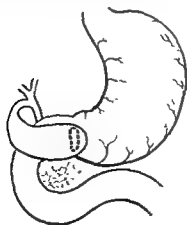


FIG. 237—Cholecyst-gastrostomy

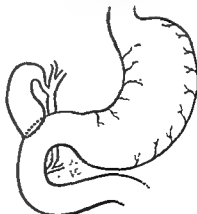


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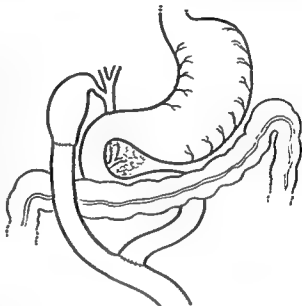


FIG. 239.—Cholecyst-jejunostomy

Risk of infection minimized

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In the two-stage procedure, at the first session the biliary obstruction is short-circuited, preferably by anastomosing the gall-bladder to the jejunum, and if thought desirable a gastro-jejunostomy may also be performed in preparation for the second stage. The technique of the second stage is similar to that of the one-stage procedure.

(a) *One-stage operation*

Many different methods of resection of the head of the pancreas have been described. The following technique is based on that of Pannett and will be found satisfactory. *Pannett's technique*

The first main step is to mobilize thoroughly the whole of the duodenum as far as the crossing of the mesenteric vessels. As a preliminary, the hepatic flexure of the colon is stripped downwards off the pancreas after dividing the peritoneal reflection. The mobilization of the duodenum, which has already been commenced as described above, is then continued, the duodenum being sponged forwards and medially along with the enclosed head of the pancreas.

The next stage is to mobilize the pylorus as in the performance of gastrectomy. The right gastric and gastro-epiploic arteries and the superior pancreaticoduodenal artery and their accompanying veins are ligated and divided. At this stage the common bile-duct can be exposed as it lies behind the first part of the duodenum, and can be divided at a suitable point above the level of the growth.

The stage is now set for division of the duodenum. Proximally it is divided in its first part—or more conveniently the stomach is divided just short of the pylorus. Distally the duodenum is divided close to the crossing of the mesenteric vessels. The distal end of the duodenum is closed and invaginated in much the same way as is the duodenal stump in gastrectomy. The remaining open ends are held in clamps, protected by sterile packs until a later stage.

All is now ready for the most crucial step in the operation—division of the neck of the pancreas at a point overlying the superior mesenteric vein. At this stage great care is required, for several small veins from the pancreas drain directly into the mesenteric vein, and they must be exposed and ligated with fine silk sutures before being cut. To do this the groove or channel which the mesenteric vein occupies behind the neck of the pancreas must be gently approached from above and below so as to separate the pancreas off the vein. When this step has been completed it only remains to dissect the unciform process of the pancreas out from between the mesenteric vein and the inferior vena cava, and the head of the pancreas can now be removed along with the duodenum.

There now lie exposed in the operative wound the cut end of the common duct (which was ligated as a temporary measure), the neck of the pancreas with the cut end of the pancreatic duct embedded in it, and the pyloric end of the stomach which has been temporarily controlled by a clamp. It remains to implant these three structures severally into the jejunum.

The jejunal loop chosen for this purpose, some 8 inches from the duodeno-jejunal flexure, is brought in front of the colon and laid from right to left across the operative field. The implantations are then carried out in turn from above downwards. The common bile-duct is anastomosed to the jejunum on its anti-mesenteric aspect by the usual four-layer suture technique. Special care is required owing to the thinness of the common duct wall, and it is important to make a watertight suture line.

In dealing with the pancreas, it is sometimes advised to implant the duct over a fine tube into the jejunum. An alternative, which on the whole is preferable, is to implant the whole thickness of the neck of the pancreas into the jejunum, thus diminishing the risk of a fistula.

This procedure is technically difficult owing to the thinness of the capsule of the pancreas, and must be performed with meticulous care to ensure a water-tight seal. The final anastomosis is made between the cut pyloric end of the stomach and the jejunum a few inches further distally. This anastomosis is made in the usual manner and presents no technical difficulty.

(b) Post-operative complications

Such an extensive operation in a debilitated patient may give rise to a considerable amount of post-operative shock. Leakage from one of the anastomoses, particularly from the bile duct, may lead to peritonitis. As in other patients with jaundice, liver failure and haemorrhage may occur. A complication of particular importance is pancreatic fistula (see p. 443). Glycosuria is rare, since a sufficiency of islet tissue is contained within the tail of the pancreas.

(7) Resection of the whole pancreas

Occasionally in extensive carcinomatous involvement it is necessary to resect the whole pancreas. The technique is similar to that employed in the partial resection described above. The additional steps involved in removal of the body and tail present no special difficulty and indeed, since implantation of the pancreatic stump is eliminated, the operation is somewhat easier. Since the splenic vessels must usually be ligated, it is necessary also to perform splenectomy.

Splenectomy

After-care

In the after-care especial attention must be paid to the carbohydrate metabolism, and insulin should be given in appropriate doses as indicated by repeated estimations of the blood sugar. After a time it is found that comparatively small doses of insulin (40–50 units daily) may suffice to maintain the equilibrium.

4. ISLET-CELL TUMOUR OF THE PANCREAS

This is nearly always a simple adenoma. It elaborates insulin and gives rise to symptoms similar to those of insulin overdosage. It is characteristic that the symptoms tend to appear during periods of fasting or after exercise, and are relieved or terminated by taking food. The symptoms may be divided into three groups: (1) disturbances of the autonomic nervous system, such as nausea and vomiting, dizziness, pallor and sweating, (2) disturbances of the central nervous system, such as convulsions with tonic and clonic spasms; and (3) psychic disturbances, such as amnesia, confusion, mania or coma. These latter symptoms may lead to a mistaken diagnosis of psychiatric disorder.

Simple adenoma

Confirmation of the diagnosis

To confirm the diagnosis an attack may be initiated by exercising the patient during a period of fasting, when the blood sugar may be reduced to as low a value as 40 milligrams per cent. The attack is quickly relieved by the intravenous administration of glucose.

(1) Operative technique

Wide exposure is necessary since the tumour may be in any part of the pancreas. Generally the tumour is of small size—rarely more than 1 centimetre in diameter—and it may be so small as to elude the most painstaking examination. It has no capsule, and is easily mistaken for normal pancreatic

tissue, from which it is distinguished only by its slightly more congested appearance and greater firmness on palpation.

The body and tail of the pancreas are inspected after tearing through the thin lesser omentum and further access is gained by division of the gastro-colic omentum. The hepatic flexure of the colon is mobilized downwards to display the anterior aspect of the head of the pancreas. The second part of the duodenum is then mobilized, by division of the peritoneal reflexion on its outer side, and sponged forwards, so that the head of the pancreas can be palpated between fingers and thumb.

If by these methods the tumour can be identified it is removed by means of a knife, along with an ellipse of adjoining healthy pancreatic tissue; the resulting haemorrhage is controlled by a few deep catgut sutures. If, on the other hand, the tumour cannot be identified, the proper course to be adopted is less clear. Generally, the wisest plan at this stage is to remove the body and tail of the pancreas up to the point at which the gland crosses in front of the superior mesenteric vein, a simple procedure when dealing with a normal gland. If naked-eye and microscopic examinations of the portion removed show no tumour and, more important, if the symptoms persist, it is necessary to submit the patient to a further operation for removal of the remainder of the gland, by a technique similar to that for carcinoma (see p. 441).

(2) Pre-operative and post-operative care

It is highly important that the patient should be brought to operation in a state of carbohydrate balance and, with this in view, the pre-operative preparation must be designed to counteract the tendency to hypoglycaemia. *Tendency to hypoglycaemia*
Purging and fasting must be avoided and normal meals taken until late evening on the day before operation. An intravenous glucose drip is then set up and maintained up to the time of, and during the course of, the exploration.

As in the case of other endocrine tumours, there is a risk that after operation the normal cells of internal secretion—the islet cells—which have been in abeyance will not at once resume their proper functions. If this happens, there is a transient danger of hyperglycaemia, which must be watched for by *Post-operative risk of hyperglycaemia*
examination of the blood and urine for sugar and controlled by prompt administration of insulin.

5. PANCREATIC FISTULA

Pancreatic fistula may follow drainage of a pancreatic cyst or occur as a complication of operations on the pancreas. Its effects vary according to the amount of secretion which escapes and its state of activation. Thus a partial loss of inactive juice is not serious, whereas a complete fistula of active juice will, if unchecked, prove rapidly fatal.

The dangerous effects are due to loss of fluids and electrolytes and to erosion of the skin and other tissues by the digestive juice. Each of these dangers requires its own special treatment. The amount of fluid lost may be as much as two litres a day, and this must be replaced by intravenous saline infusions. Plasma or whole-blood transfusions may be required, and the nutrition should be maintained by administration of protein hydrolysate.

To prevent skin erosion, a fine rubber catheter should be inserted into the fistula and connected with a constant-suction pump to divert the juice. A

rubber pessary affixed to the skin by rubber solution encloses the fistulous area and limits any leakage. Alternatively, the catheter may be inserted through a sponge-rubber pad which, when applied to the skin, is held in firm contact through suction. If the patient can be turned prone he may be nursed on a ventral plaster with a gap opposite the fistula so that the escaping fluid drains directly away from the skin surface. The use of ointments, such as that of kaolin in lanolin or aluminium paste, has also been recommended.

Usually, if the patient's general condition can be maintained, the fistula tends to close spontaneously. If not, it may be necessary to dissect out the fistula and implant it into a jejunal loop. The administration of atropine, large doses of sodium bicarbonate by mouth, and a high fat and protein diet, have been recommended with the object of diminishing the pancreatic secretion.

If a pancreatic fistula shows no signs of closing after six weeks it is important to discover whether the leak is occurring from the main duct, and further to determine whether there is obstruction to the flow of pancreatic juice along this duct into the duodenum. Information may be obtained from skiagrams after the injection of Lipiodol into the fistula. If the main duct is seen to be patent into the duodenum the fistula should ultimately close spontaneously; if the main duct is obstructed, implantation of the fistula into the intestine will be necessary.

REFERENCES

Opie, E. L. (1901). *Bull. Johns Hopk. Hosp.*, 12, 182.

[References to other titles are given under *Pancreas* in the Index Volume. The subject of *Pancreas, Diseases*, is also dealt with in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 386.]

PARALYSIS—MANAGEMENT OF

By L. GUTTMANN, M.D., M.R.C.P.

NEUROLOGICAL SURGEON-IN-CHARGE, HEAD AND SPINAL INJURY CENTRE,
MINISTRY OF PENSIONS' HOSPITAL, STOKE MANDEVILLE

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1. INTRODUCTION AND DEFINITIONS

258.] This article deals with the management of paralysis of the central nervous system, especially the spinal cord, as in paraplegia, hemiplegia or paraplegia. Such paralysis is of varying magnitude, and generally involves such essentials as motor, bladder, intestinal and sexual functions.

In the past, persons with such severe lesions were helpless cripples, and the patient with a complete lesion of the

restored to a useful life.

2. NUTRITION

The maintenance of nutrition at the highest possible level is of prime importance, particularly in cases of paralysis complicated by

to infection of the urinary tract and pressure sores, when there is a continuous loss of protein. This can result in extreme degrees of malnutrition (Figs. 241 and 242) comparable to those found in patients in concentration camps.

To combat nutritional deficiency and for the rapid restoration and maintenance of satisfactory nutritional conditions, blood transfusions have proved most beneficial in these cases, and time should not be wasted by trying these patients first with iron and liver, as the effect of these is uncertain and slow.



FIG. 241.—Patient with gunshot injury to the spine, with complete transverse lesion at Th 10. Profound emaciation on admission to Spinal Centre, nine months after injury.

The author considers that in paralysed patients with signs of nutritional deficiency and septic conditions, due especially to urinary infection, blood transfusions are imperative, even if the blood count does not show a severe degree of anaemia. It may be noted that, in these cases, the average value of haemoglobin varies between 65 per cent and 78 per cent, and erythrocytes vary between 3 million and 3.8 million. Moreover, as vomiting is common in these cases, the blood count may show normal values, owing to haemoconcentration. Blood transfusion is not contra-indicated in cases of increased blood urea, indeed, it has been demonstrated repeatedly that blood transfusions have the effect of diminishing the amount of blood urea, especially in cases

with noticeable degrees of anaemia. Whenever possible, fresh blood should be transfused. When vomiting, diarrhoea or sweating causes dehydration, saline and glucose drips are essential for safeguarding the water and chloride balance of the body.

Once the acute stage of nutritional deficiency is past, a special diet, rich in protein and vitamins, supplemented in certain cases by hydrolysed protein such as Pronutrin, combined with liver-extract injections and iron has proved effective. Constant attention must also be directed to the maintenance of good nutrition after discharge from hospital in those patients with bladder paralysis and chronic infection of the urinary tract. A high protein diet is recommended, even though the protein level in the blood may be normal. Experience has shown that the assessment of plasma protein is not a reliable

Blood
transfusions

Measures
against
dehydration

High
protein diet



FIG 242—Same patient as in Fig 241. (a) five weeks later, (b) thirteen weeks later

guide to the degree of malnutrition, for a person with such an extreme degree of emaciation as that shown in Fig. 24I may show a normal or only slightly diminished value.

Another important point in the diet of paralysed patients, who are long-term institutional cases, is the prevention of monotony. There is no doubt that the maintenance of a good morale of these patients depends, to a large extent, on the ingenuity of the people concerned with the patients' dietary needs.

3. CARE OF THE SKIN

This is of cardinal importance after acute spinal cord lesions especially in the early stages. Traumatic shock and paralysis of the spinal vasomotor centre result in loss of tone and of vasomotor control; these cause tissue resistance to pressure to be lowered in the insensitive parts of the body. Therefore, pressure easily produces ischaemia, which may progress into bedsores. Details of the prevention and treatment of bedsores have already been described by the author (Vol. 2, p. 65).

From the beginning, every effort should be made to restore vasomotor control and promote good circulation. Although the vasomotor control in the paralysed areas gradually recovers, it is immediately grossly impaired by any intervening acute infection, and by post-operative shock following subsequent major surgical procedures. Therefore, during these periods, in order to prevent damage to the skin and deeper tissues, the intervals between turnings should be shortened.

Vasomotor disturbances can, however, be permanent, and occur especially in the distal parts of the paralysed limbs, as shown by the pinkish-blue colour of the cold toes and feet in cauda equina lesions and following poliomyelitis. These disturbances increase in wet and cold weather, and precautions should be taken to keep the feet and legs warm by wearing woollen socks and boots such as flying-boots, and by preventing the legs from being kept for too long in one position. Hot-water bottles should never be used for paraplegics.

The skin should be kept scrupulously clean by frequent washing with soap and water, to facilitate efficient cutaneous function. It has been found that the feet, especially the soles, are often neglected. Indeed, the degree of scalliness of the soles and heels is an excellent indicator of the efficiency of the medical and nursing staff concerned with the management of paralysed patients. The vital importance of an hygienic condition of the body, especially in those areas of the skin which are easily saturated with urine and faecal matter, should be explained to the patient from the beginning, and he should be trained as soon as possible to carry out the cleaning.

Outbursts of profuse sweating over both the paralysed and the normal parts of the body occur in certain stages after spinal cord injury. This condition can be extremely distressing and, like flexor spasms, can make the patient's life most miserable. It represents part of the mass response of autonomic reflex mechanisms, and can be elicited by any extrinsic or intrinsic factor in the paralysed part of the body (Head and Riddoch, 1917; Guttman, 1946; Guttman and Whitteridge, 1947). The main factor in eliciting outbursts of sweating is stagnation in the urinary tract or colon, leading to distension of these viscera. The frequency of this reflex response, when due to bladder

*Vasomotor
paralysis*

*Restoration
of vasomotor
control*

*Profuse
sweating*

distension, depends largely on the capacity of the bladder, and it is especially marked in cord lesions in which the bladder has become contracted. The recognition of this reflex syndrome by medical and nursing staffs is a valuable guide for immediate and correct action. Whatever the cause of profuse sweating the continuous loss of chlorides through the sweat glands can be serious, and it has to be replaced by saline administered either by mouth or by intravenous drips.

4. CARE OF THE BLADDER

Neglect and inadequate treatment of the bladder are the commonest causes of death of paralysed patients.

Certain investigations of the urinary tract should be used as a routine and at regular intervals. These are: (1) bacteriological examination of the urine; (2) blood urea; (3) skiagrams of the bladder and kidneys; (4) cystogram; (5) intravenous and, if necessary, retrograde pyelograms; (6) cystometro-gram; and (7) residual urine test. *Methods of investigation*

Sometimes special functional tests, such as urea concentration test, indigo-carmine test, phenol-sulphone-phthalein test, and blood-urea clearance test may be necessary, and usually—especially just prior to closure of a suprapubic cystotomy—cystoscopy is indispensable. In patients with closed bladders and a large amount of residual urine, cystoscopy is necessary to ascertain whether this is due to hypertrophy of the bladder neck.

The stages of bladder paralysis which inevitably follow severe lesions of the spinal cord or cauda equina can be classified as follows. (1) Complete retention; (2) passive incontinence, due to overflow from the distended bladder; and (3) periodic micturition either (a) by reflex activity of the automatic bladder, or (b) by expressing the urine, using the muscles of the abdominal wall. This can be supplemented by pressing the lower abdomen with the hand. The latter type of periodic micturition is the rule in lesions involving the spinal bladder centres or the roots of the cauda equina which connect the bladder to its spinal centres, producing what is known as an autonomous bladder. *Classification of bladder paralysis*

(1) Aims of treatment

The main aims in the care of the bladder are: (a) prevention of direct damage to the bladder and urethra by instrumentation and artificial drainage; (b) prevention or treatment of ascending infection of the urinary tract; (c) maintenance or restoration of an adequate bladder capacity; and (d) restoration of efficient micturition by normal channels, with the least possible degree of residual urine or of incontinence.

Opinions still differ as to the most suitable methods of achieving these aims, but, as the result of experience gained in treating over 400 patients with bladder paralysis during recent years, the author has come to the following conclusions.

(2) Initial treatment of paralysed bladder

As a rule, the paralysed bladder is never so distended after an acute spinal cord lesion as to warrant immediate drainage by any method. Therefore, the bladder can be safely left alone for 24 hours or longer, unless distension produces unbearable pain or sets up distress due to reflex responses of autonomic *No immediate drainage*

mechanisms (Guttmann and Whitteridge, 1947). During this period, if the acute spinal cord lesion is caused by injury, every effort should be made to combat traumatic shock by blood transfusions and other means, and thereafter to move the patient to a hospital, preferably a spinal centre equipped with all facilities for efficient handling of such a patient. In war-time, spinal injuries deserve highest priority for evacuation by air. In exceptional cases, aspiration of the bladder through a serum needle inserted well above the pubis may be permissible, if surgical facilities are poor for performing bladder drainage by urethral catheterization or suprapubic cystotomy. Manual expression of the distended, atonic bladder is hazardous.

(3) Drainage of the bladder

If voluntary or reflex micturition has not developed within 24 to 48 hours, permanent drainage of the bladder is indicated. Three methods have been used for drainage.

- (a) Urethral catheterization (intermittent or continuous).
- (b) Suprapubic cystotomy (high and low).
- (c) Urethrostomy.

From experience gained, it can now be concluded that: (i) neither urethrostomy nor low suprapubic cystotomy should ever be used; (ii) neither of the other methods has proved to be a safeguard against ascending urinary infection or the method of choice for every case of paralysed bladder; certain circumstances may make one preferable to the other during the various stages of bladder paralysis; and (iii) whatever method of bladder drainage is used, it should be combined with systemic courses of sulphadiazine or Sulphamezathine (1 gramme three times daily), and penicillin (50,000 units 3-hourly) from the start.

(a) Urethral catheterization

Unless, for special reasons (see p. 452), high suprapubic cystotomy is indicated, the author considers that urethral catheterization should be the method employed for bladder drainage. Any medical officer concerned with the treatment of paralysed patients must be absolutely familiar with a "non-touch" technique of urethral catheterization, particularly as this is also the method used in later stages, especially following closure of suprapubic cystotomy. Inexperienced persons, such as orderlies, should never be allowed to carry out urethral catheterization. In certain circumstances, the author has found it safer, in the later stages, to teach the patient to catheterize himself, under scrupulously aseptic conditions.

Procedure.—For drainage of the bladder following spinal cord injury, the author finds the following procedure satisfactory.

Intermittent catheterization should be performed every 8–12 hours for the first few days. The reason for first using intermittent catheterization and not an indwelling catheter is to allow the urethral mucosa to become accustomed gradually to the foreign body. Moreover, in cases with incomplete lesions in which early return of bladder function can be expected, intermittent catheterization allows distension of the bladder, which is a strong stimulus for promoting the return of bladder activity. The "non-touch" technique of urethral catheterization is as follows.

Combat of traumatic shock

Immediate transfer to spinal centre

Aspiration of bladder

"Non-touch" technique

First stage Intermittent catheterization

(i) The surgeon, wearing a mask, retracts the prepuce and wraps the penis *Technique* in a piece of gauze soaked in Dettol solution, leaving only the glans free.

(ii) Thorough cleansing of the glans with swabs soaked in Dettol solution, using forceps.

(iii) The penis is supported in an upright position, so that the glans does not touch anything. The areas above and below the penis are covered with a sterile towel.

(iv) If an assistant is available, he now removes the sterile catheter from its sterile container, with the forceps reserved for this purpose, and hands it to the surgeon, who, receiving it with fresh, sterile forceps, lubricates the proximal end by drawing it through oxycyanide jelly on a sterile gauze and introduces the proximal end into the urethra, with the aid of the forceps. Only soft rubber catheters of small size (Jaques 6-8) should be used.

(v) At frequent intervals a urine specimen is sent for bacteriological investigation.

After 4-8 days, or once the urine has become infected (this is unavoidable *Second stage* in time, whatever the form of artificial drainage used), an indwelling catheter, *Indwelling catheter* combined with tidal drainage (see p. 455), is the method of choice. However, it is essential that the indwelling catheter be changed every other day at first,

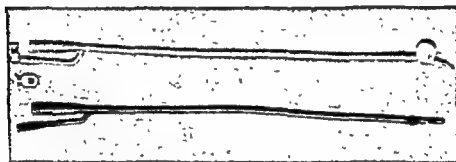


FIG. 243.—Foley catheter with 5-cc. balloon (one unfilled, the other filled with 5 cc. sterile water and clamped, as it would appear *in situ*, after urethral catheterization or suprapubic cystostomy).

and later on at intervals of 2-3 days. In order to free the urethra of deposits before inserting the new indwelling catheter, the urethra itself should be washed out with Flavazole, 1 : 2,000, using a small tube fixed on to a 20-cubic-centimetre syringe. Such washouts are especially necessary when urethritis *Urethritis* has developed. Flavazole has also proved successful in the treat- *Balanitis* ment of balanitis, which is liable to develop beneath a long foreskin. Early circumcision is indicated in such cases. The best type of indwelling catheter is the self-retaining Foley catheter of small size (5-cubic-centimetre balloon, Fig. 243).

Once the automatic function of the bladder has returned, which in uncomplicated transverse lesions of the spinal cord usually occurs after 5-8 weeks, the indwelling catheter should be removed, and intermittent catheterization must be continued until detrusor action is powerful enough to empty the bladder or leave only a trace of residual urine. The same principle applies to cauda equina lesions, when voluntary micturition is possible by pressure on the abdominal wall. *Third stage* *After-care*

*Indications**Emergency
method under
battlefield
conditions**(b) Suprapubic cystotomy: first stage*

In the early stages of bladder paralysis when the injury is associated with local damage to the urethra, with urethral stricture, or with gonorrhoeal infection, suprapubic drainage is indicated in preference to urethral catheterization. In war-time, under battlefield conditions, suprapubic cystotomy represents a useful emergency method for the early treatment of the paralysed bladder, if immediate transfer from one hospital to another is unavoidable and frequent transfer by air to more suitable surroundings is impossible.

(i) *Technique of open method.*—The opening of the suprapubic wound should lie half-way between the pubis and umbilicus, and a small suprapubic catheter should be inserted into the upper part of the distended bladder, in an oblique, downwards direction. This procedure prevents such maximal constrictures of the bladder and adherence of the tract to the pubis as are seen regularly following low suprapubic cystotomy. As a rule, the first change of catheter should be made after 8–10 days, as this gives ample time for the formation of a good, oblique track. Subsequently the catheter, preferably a straight one fixed to a rubber shield, should be changed once a week, under scrupulously aseptic conditions. If a self-retaining catheter of the Malecot or De Pezzer type (which should never exceed size 24) is used, its removal, as well as its introduction, should be carried out with great caution and gentleness, to prevent damage or perforation of the bladder wall. The practice of pulling such a catheter out briskly, without having straightened its proximal, self-retaining end with an introducer, is most harmful. It is much better to avoid Malecot and De Pezzer catheters altogether and employ self-retaining catheters of the Foley type (5–20-cubic-centimetre balloon) which have the great advantage of being introduced as a straight catheter without the aid of an introducer before the balloon is expanded.

(ii) *Technique of closed methods.*—A trocar has sometimes been employed for suprapubic drainage of the distended bladder. The method adopted for this procedure is either Kidd's, using a trocar and cannula designed for the introduction of a self-retaining catheter of a larger size, or Riches's new method of suprapubic catheterization (Riches, 1943), employing a very small catheter (size 16F) fixed to a trocar. There are at present no comparative data available which would allow a definite decision as to whether Riches's method is superior to the older trocar methods, or whether closed suprapubic catheterization is preferable to open cystotomy. Whatever closed method is used, care has to be taken that the trocar is inserted well above the pubis in the midline and that the bladder is maximally distended. As a rule, this precaution diminishes the risk of injury to the peritoneum, because, during bladder distension the peritoneum is raised, and thus an uncovered area of the bladder is exposed, through which it is safe to push the trocar into the bladder. However, in certain cases, as observed on several occasions by Macalpine (1948), the peritoneum may be anchored down to the pubis, forming a pouch in front of the distended bladder, which may even contain small gut. In such cases, the danger of causing peritonitis with any closed method is great, particularly if the distended, paralysed bladder is already infected, either spontaneously or by previous urethral catheterization. It is essential that the catheter be firmly fixed to the trocar, otherwise it may break loose before entering the bladder. Once the catheter is inserted and suprapubic drainage is established,

*Danger of
peritonitis*

care has also to be taken not to pull out the catheter on turning the patient during the first few days following this procedure, for, if a small catheter has been used, it may be difficult or even impossible to insert a new catheter through the very small opening.

(c) *Suprapubic cystotomy: second stage*

In later stages of bladder paralysis, suprapubic cystotomy may be indicated *Indications* to allow the healing of urethral, scrotal and perineal fistulas, and also when severe infection of the bladder leading to frequent pyrexial attacks cannot



FIG. 244—Case of complete transverse lesion at Th 12, with immediate suprapubic cystotomy following injury (a) Bilateral hydronephrosis with contracted bladder (b)

*No safeguard
against
ascending
infection*

be controlled by urethral catheterization. However, suprapubic cystotomy has not proved to be a safeguard against ascending urinary infection, stone formation in bladder, ureters and kidneys, pyonephrosis or hydronephrosis. The danger of transferring virulent organisms via the suprapubic catheter through the bladder and open ureters directly into the pelvis and calyces is considerable, in view of the fact that reflux into the ureters and kidneys is often revealed by cystograms. In middle and upper thoracic, as well as in cervical and certain cauda equina lesions, the bladder has often been found to be contracted. In several of these cases, extreme degrees of hydro-ureter and hydronephrosis were found (Fig. 244), in spite of the fact that cystoscopy did not reveal blockage of ureteric orifices. This observation is at variance with the opinion held generally that the only source of hydro-ureter and hydronephrosis is back pressure from the dilated bladder. In the author's opinion, the ascending infection itself is just as important as the back pressure.

*Contracture of
bladder*

*Hydro-
nephrosis*

*Early
discontinuation*

Therefore one should aim at discontinuing suprapubic drainage at the earliest possible moment, and if this can be within the first few weeks or months after injury it is the author's experience that urethral micturition may be re-established and infection of the urinary tract may be overcome. In cases in which suprapubic drainage has been continued for longer periods, healing of the suprapubic wound is always delayed (*see also* Talbot, 1946).

(d) Suprapubic cystotomy: third stage

After-care

When suprapubic drainage is discontinued, it is necessary, during the initial period, to carry out temporary drainage with an indwelling catheter of small size, which should be changed every other day. Afterwards, intermittent catheterization at various intervals will be necessary in many cases in order to measure the residual urine, to remove deposits and to combat infection by regular washouts. This can be done at home, after the patient's discharge from hospital, by any practitioner who is familiar with a "non-touch" technique of urethral catheterization. However, in certain cases, especially those with quiescent renal stones and those with hydronephrosis, readmission to hospital for clinical review will be necessary at regular intervals.

*Important
symptoms of
active
infection of
urinary tract*

A most important point in the after-care of the closed, paralysed bladder, regardless of whether the initial treatment was carried out by urethral catheterization or suprapubic cystotomy, is to make the patient conscious of certain symptoms indicating active infection of the urinary tract, which require immediate medical attention. These are loss of appetite, feeling "seedy and off colour", loss of weight, any discomfort in the abdominal region (referred to often by the patient as "stomach trouble" or "feeling liverish"), and headache, and they can be present when there is purulent urine in the bladder long before the rigor or pyrexia of the acute attack. Inflammation of the centre of the suprapubic scar may be another early sign of active cystitis, and an indication for immediate urethral catheterization and irrigation of the bladder.

(4) Control of urinary infection

(a) Avoidance of recumbency and restoration of physical fitness

Every endeavour should be made to avoid prolonged recumbency by encouraging all forms of movement, even in the first stages after a spinal cord lesion. This is of vital importance in preventing stagnation of urine, which

encourages infection and leads to the formation of stones in the kidneys and bladder. Any form of fixation in a plaster cast or plaster bed is strongly deprecated. The combating of nutritional deficiency and the restoration of physical fitness are also of importance in controlling bladder infection.

(b) *Irrigation of bladder and kidneys*

The intake of a sufficient amount of fluid, especially between meals, is the best method of irrigating the kidneys. It can be taken in the form of tea, or barley water which can be mixed with fruit juice to make it more palatable. As a rule, a paraplegic should drink at least five or six pints in 24 hours, and even this amount should be increased during attacks of pyelitis. *Fluid intake*

In cases with suprapubic drainage or indwelling catheterization, intermittent irrigation of the bladder should be carried out at least once a day, especially in the morning. In every case the amount of fluid which the bladder can hold without overdistension should be ascertained. Thorough cleansing of the bottom of the bladder and of diverticula requires the use of a bladder syringe rather than a funnel. Solutions should be tepid (90°–98° F.). The fluid recommended for irrigation, when the urine is alkaline, is $\frac{1}{2}$ per cent acetic acid, oxycyanide of mercury (1 : 4,000), 4 per cent boric acid, or Flavazole (1 : 2,000). When the urine is acid, normal saline, silver nitrate (1 : 2,000), potassium permanganate (1 : 2,000), or Flavazole (1 : 2,000) can be used. If the urine is infected with *Bacillus pyocyaneus*, washouts with phenoxetol 2.4 per cent, followed by saline, can be used for 2–3 days. The efficiency of these irrigations is often increased by frequent changes in the solutions employed, and in the pH of the urine, an effect which can be intensified by administering 30 or more grains of acid sodium phosphate, given three or four times a day, by mouth. Intolerance to this drug is indicated by diarrhoea. Acid sodium phosphate may be replaced by 10–30 grains of ammonium benzoate in stearate or glutoid capsules, or by mandelic acid. In cases with phosphatic deposits, solution G (Suby, Suby and Albright, 1942) should be used for irrigation. *Local irrigation*

Tidal drainage has proved to be a valuable auxiliary method, with either suprapubic drainage or indwelling urethral catheterization, especially in the early stages. It is of special value in removing mucus and small stones from the bladder, particularly from the posterior wall when the patient has been lying prone. Various designs of tidal-drainage apparatus have been recommended (Laver, 1917; Munro, 1943; Lawrie and Nathan, 1939; Riches, 1943; Stewart, 1942). The author has found Riches's apparatus very satisfactory, and, being fitted with a manometer, it can also be used for cystometrograms. However, tidal drainage is of use only if it is understood by all concerned, including the patient, and is constantly supervised. In certain cases of small bladders with uninhibited detrusor activity in cord lesions, and also in some cases of cauda equina lesions, with rigid bladder walls, tidal drainage has not worked satisfactorily, in spite of all efforts. *Tidal drainage*

(5) *Chemotherapy*

When an acidifying agent, such as acid sodium phosphate, ammonium chloride, ammonium benzoate or mandelic acid, is being given, hexamine (10 grains three times daily) has at least an inhibitory effect on bacterial growth, by liberating formaldehyde. However, larger doses may give rise to

gastro-intestinal disturbances and haematuria. In pyelitis, the author has sometimes found intravenous injections of 5 cubic centimetres of Cylotropin (combination of hexamine, sodium salicylate and caffeine) once or twice daily to be effective and superior to other forms of chemotherapy.

The discovery of the sulphonamides, penicillin and streptomycin has undoubtedly increased our facilities for effective combat of infection of the urinary tract and has made possible a more specific treatment of various types of organism. Of the sulphonamides, sulphadiazine and Sulphamezathine have proved more suitable than sulphathiazole in pyelitis. Whereas the sulphonamides have been used in the treatment of both Gram-positive and Gram-negative organisms, penicillin has been found more effective against Gram-positive organisms, especially the haemolytic streptococcus. The author's routine treatment of pyelitis consists of combined courses of sulphadiazine or Sulphamezathine, starting with 2 grammes and continuing with 1 gramme every four hours, up to 20–35 grammes, and intramuscular injections of penicillin 30,000–50,000 units 3-hourly, up to a total of 2,000,000–3,000,000 units.

More recently, streptomycin has proved to be effective against urinary infection caused by Gram-negative or mixed Gram-positive and Gram-negative organisms, especially in those cases which do not require instrumentation and catheterization (personal observation). Catheterization during or following treatment may result in drug-resistant infections (Petroff and Lucas, 1946; Hewitt, 1947; Pulaski, 1946). Administration of this drug for pyelitis has resulted in dramatic clinical improvement, with decrease of leucocytosis and improvement of renal function. Streptomycin has also proved of value in the preparation of patients for surgical procedures. The antibacterial effect of this drug increases if the pH of the urine is more than 7.0 (Abraham and Duthie, 1946; Wolinsky and Sirecken, 1946). The following conditions have been found to be unfavourable to the use of streptomycin. (1) Obstruction in the urinary tract; (2) foreign bodies, such as calculi and indwelling catheters; (3) wounds with granulating surfaces; and (4) undrained abscesses.

Limitation of fluid intake to 2,500 cubic centimetres daily and a dose of 1 gramme of streptomycin per day was found sufficient to guarantee a concentration of 100 micrograms per cubic centimetre of urine, but doses of 2.5–2.6 grammes given in 0.5–1.0 gramme amounts every 4–6 hours have been advocated. An initial dose of 0.5–1.0 gramme in 20 cubic centimetres of saline solution can be given intravenously. Intramuscular injections are given in 4.5 cubic centimetres of saline solution.

(6) Stone formation

In paraplegics, calculi usually form as a result of infection. Stagnation of urine, particularly through recumbency and immobilization, and kink formation in the ureters are the most important factors in promoting stone formation. If there is a suprapubic catheter, stones usually form around it (Fig. 245), but they are also not uncommon in closed bladders (Fig. 246). Calculi in paraplegics consist almost entirely of phosphates. Once formed, they show little or no tendency to disappear when the patient is able to get up, nor can they be dissolved easily and quickly by agents such as solution G (Suby, Suby and Albright, 1942). In most instances they need surgical removal, as

Sulphamezathine in pyelitis

FIG. 245.—Stone formation in bladder, following suprapubic cystostomy



FIG. 246.—Stone formation in closed bladder.

*Surgical
methods of
choice*

early as possible, particularly if there is chronic infection with febrile attacks. Only small quiescent stones, limited to the cortical part of the kidney, or small mobile stones in the renal pelvis may be treated conservatively. Nephrostomy and pyelolithotomy are the methods of choice, and nephrectomy is indicated only if it is proved, beyond all doubt, that the kidney has lost its secretory function and is the cause of general infection. After removal of renal stones, frequent irrigation of the kidney with solution G through the nephrostomy tube is recommended to dissolve residual fragments, and an early return to activity is necessary to prevent the recurrence of stones.

(7) Hydronephrosis

*Effect of
pituirrin*

It is beyond the scope of this article to discuss the mechanism and treatment of various types of hydronephrosis which may result from complications in the urinary tract, following transverse lesions of the spinal cord. But it is worth while mentioning that, in certain cases of marked hydronephrosis, without raised blood urea, pituitrin has been shown to have a remarkably beneficial effect on urinary function. This has been described by Bell (1947) in cases of hydronephrosis with or without paraplegia. The author has confirmed these findings in a few selected cases of paraplegia with marked hydronephrosis and hydro-ureter. Cystograms taken before pituitrin treatment in a complete transverse lesion at Th.4 showed gross unilateral hydronephrosis on the left side. After daily injection of 0.5 cubic centimetre of pituitrin for a fortnight, a decrease in the size of the kidney and ureter was apparent, and after further treatment for 9 days the cystogram showed almost normal conditions. Examination after 3 months, in this case, still showed normal conditions although the treatment had been discontinued for over 2 months. While it is rather premature to draw final conclusions, the results so far achieved are encouraging.

(8) Retraining of the bladder

Repeated cystometrograms have proved invaluable in ascertaining the func-

temporary plugging of the cystotomy tube is most important for retraining of the bladder.

*otions
ated
bladder
vision*

Most paraplegics can be trained to an awareness of the degree of fullness of the bladder, by appreciation of certain sensations associated with bladder distension. These sensations are pain, burning, quivering or just pressure, and may be referred to the bladder itself, the suprapubic region, the root of the penis, the penile urethra or the lateral aspect of the thighs. In complete lesions of the upper thoracic and cervical cord, bladder distension sets up certain reflex responses of autonomic mechanisms (Guttmann, 1947), which are recognized by an ascending sensation in the midline of the body, shivering, hot feeling in face and neck, blockage of nasal air passage, head fullness, headaches in the back of the head, slowing of pulse, extrasystoles and sweating.

The patient gradually learns to urinate reflexly by employing certain trick
middle
rapubic
region, or inserting a finger into the anus. In conus and cauda equina lesions,

*anism
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rition*

voiding is achieved by applying abdominal pressure. It is obvious that, in these cases, the more the abdominal muscles are trained and over-developed, the more satisfactory the voluntary micturition.

In many cases, especially in those following discontinuance of suprapubic drainage, the voiding mechanism of the bladder is imperfect and residual urine results. If this is small in amount (under 3 ounces), with a bladder capacity of at least 8-10 ounces, there is no harm, so long as stagnation of urine in the bladder is avoided by a high fluid intake. Carbachol may sometimes be useful in promoting return of voluntary or reflex micturition. Repeated estimations of residual urine are necessary, and if a large amount of residual urine is due to obstruction of the bladder neck, caused by hypertrophy and fibrosis of the tissues of the internal meatus, transurethral resection of the bladder neck is required (Emmett, 1943 and 1947; Thompson, 1945). This operation may have to be repeated to produce appreciable diminution of residual urine and facilitate voluntary or reflex micturition. The danger of the operation is post-operative haemorrhage, which must be treated without delay because of the disastrous consequences of blood loss in a paralysed patient.

Control of residual urine

Transurethral resection of bladder neck

Post-operative haemorrhage

Incontinence of urine can be overcome by training the patient to adjust the frequency of his micturition to the amount of fluid intake. However, most patients, especially those with distal cauda equina lesions, need a urinal, as sudden movements and acute increase of abdominal pressure by coughing and sneezing may result in incontinence of a few drops or more of urine. As a rule, the person with a paralysed bladder should be provided with two urinals for hygienic purposes. In order to prevent reflux of urine from the urinal into the trousers, which usually occurs when the patient is seated in his chair, the author has designed a horseshoe-shaped Sorbo cushion, which slopes towards the front, so that the urinal and penis lie in a downward direction.

Control of incontinence

5. BOWEL FUNCTION

In the early stage of an acute transverse spinal lesion, intramuscular injection of Prostigmin 5-10 milligram or 1 cubic centimetre of pituitrin, every 4-7 hours, has proved beneficial in overcoming the intestinal paralysis. Furthermore, for relief of abdominal distension, due to intestinal paralysis, the passing of a rectal tube has proved invaluable. In dealing with paraplegics in later stages, it must always be remembered that bowel action may be due merely to overflow. Such patients will state that they have regular motions, while rectal and abdominal investigations reveal masses of old and hard faeces, which lead to overdistension of the colon and set up reflex responses of skeletal muscles and the cardiovascular system, as mentioned above. Regular enemas are required, drugs such as cascara and senna and liquid paraffin are useful, and attention must be paid to the diet.

*Constipation
Bowel action due to overflow*

Continuous overloading of rectum and sigmoid may also lead to obstruction of the venous return from the great pelvic veins, which may result in oedema of the legs. The author has, on occasions, observed that the oedema has disappeared once the overloaded rectum has been evacuated and that it will not recur so long as the rectum is kept free. Profuse diarrhoea in paraplegics needs greatest attention, for this, as well as profuse sweating, may indicate

Oedema of legs due to venous obstruction

Diarrhoea and renal deficiency

*Surgical
methods of
choice*

early as possible, particularly if there is chronic infection with febrile attacks. Only small quiescent stones, limited to the cortical part of the kidney, or small mobile stones in the renal pelvis may be treated conservatively. Nephrostomy and pyelolithotomy are the methods of choice, and nephrectomy is indicated only if it is proved, beyond all doubt, that the kidney has lost its secretory function and is the cause of general infection. After removal of renal stones, frequent irrigation of the kidney with solution G through the nephrostomy tube is recommended to dissolve residual fragments, and an early return to activity is necessary to prevent the recurrence of stones.

(7) Hydronephrosis

*Effect of
pituitrin*

It is beyond the scope of this article to discuss the mechanism and treatment of various types of hydronephrosis which may result from complications in the urinary tract, following transverse lesions of the spinal cord. But it is worth while mentioning that, in certain cases of marked hydronephrosis, without raised blood urea, pituitrin has been shown to have a remarkably beneficial effect on urinary function. This has been described by Bell (1947) in cases of hydronephrosis with or without paraplegia. The author has confirmed these findings in a few selected cases of paraplegia with marked hydronephrosis and hydro-ureter. Cystograms taken before pituitrin treatment in a complete transverse lesion at Th.4 showed gross unilateral hydronephrosis on the left side. After daily injection of 0.5 cubic centimetre of pituitrin for a fortnight, a decrease in the size of the kidney and ureter was apparent, and after further treatment for 9 days the cystogram showed almost normal conditions. Examination after 3 months, in this case, still showed normal conditions although the treatment had been discontinued for over 2 months. While it is rather premature to draw final conclusions, the results so far achieved are encouraging.

(8) Retraining of the bladder

Repeated cystometrograms have proved invaluable in ascertaining the functional state of the bladder, in determining the various stages in the return of bladder activity and in bladder retraining. In cases with suprapubic drainage, temporary plugging of the cystotomy tube is most important for retraining of the bladder.

*Sensations
associated
with bladder
distension*

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*Trick
mechanisms
for starting
micturition*

mechanisms for starting micturition, such as rubbing the glans or the lateral aspect of the thigh, gently patting, rubbing or squeezing the suprapubic region, or inserting a finger into the anus. In conus and cauda equina lesions,

voiding is achieved by applying abdominal pressure. It is obvious that, in these cases, the more the abdominal muscles are trained and over-developed, the more satisfactory the voluntary micturition.

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*Constipation
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Obstruction of legs due to venous return

Diarrhoea and sweating

renal deficiency, and no time should be lost in administering saline intravenously, until the kidneys have recovered.

6. SEXUAL FUNCTION

There is widespread belief that

that this is not the case, and in a number of patients sexual readjustment is

possible to varying degrees. It is beyond the scope of this article to go into all details of this problem, but a few important points can be mentioned. It must be remembered that, in a transverse lesion of the spinal cord, the erection reflex is one component of the reflex activities below the level of the lesion which return once the spinal shock has subsided. It has been observed that, by appropriate local stimuli, the erection reflex can be utilized for intercourse in certain cases of paraplegia. It is true that, in cases with marked spasticity, flexor and particularly adductor spasms may render intercourse impossible, but then the denervation of the adductors by neurectomy of the obturator nerves must be considered. Activity of the reproductive organs at the moment of ejaculation of seminal fluid sets up certain reflex responses and sensations similar to those found on bladder distension. Therefore, certain paralysed patients can be trained to become aware when ejaculation occurs during intercourse. The fertility in paralysed persons can be ascertained by an intrathecal Prostigmin test, discovered recently by the author (Guttmann, 1947). While Prostigmin (0.3–1.0 milligram) injected intrathecally acts as a depressant on the skeletal muscles (Kremer, 1942), it has a stimulating effect on the reproductive organs. Therefore, this method can be used for investigating the chances (including the possibilities of artificial insemination) of reproductive activities in a person with a paralysis of any kind.

Utilizing of erection reflex

Measures to deal with spasticity

Reflex responses and sensations due to ejaculation

Effects of Prostigmin on reproductive activity

7. PHYSICAL RESTORATION

Physiotherapy in paralysed persons, to be effective in restoring the patient's fitness for independence, working capacity and family life to the highest possible degree, must be applied early and practised persistently. The aims of physiotherapy can be divided into three main groups.

Early start

Correct position of paralysed limbs

(1) Prevention of contractures and of atrophy

The methods employed include keeping the limbs in the correct position, passive movements, bath therapy and surgical procedures. In the early stages the paralysed limbs must be kept in a position to prevent adduction of the legs, flexion of the knees and hips, drop-foot, claw

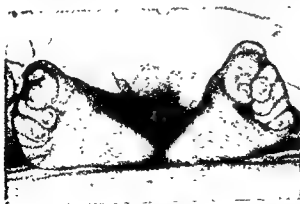


FIG. 247.—Flexion contracture of toes, due to neglect, in a case of cauda equina lesion.

injection of 10–15 cubic centimetres of absolute alcohol made at the interspace between the first and second lumbar vertebrae, may be tried first, as being the less serious undertaking, especially in cases in which the poor general condition of the patient makes a big operation a greater risk (Guttmann, 1947; Gingras, 1948). The immediate effect of such an injection in

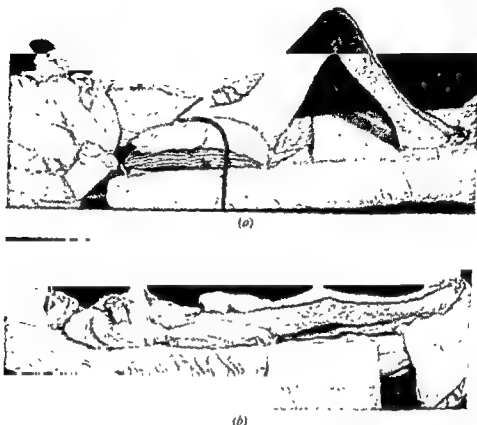


FIG. 249—Flexor spasm (a) before and (b) after intramedullary alcohol injection

abolishing the most violent flexor spasms can be very striking, as shown in a case of complete lesion at Th.10 (Fig. 249). However, experience has shown that the alcohol injection may have only a temporary effect.

(3) Adaptation by compensatory training of normal parts of the body

Exercises of normal parts of the body are carried out, with a view to readjustment of the vasomotor control to postural changes (particularly in upper thoracic and cervical cord lesions) and to the over-development of those muscles which are essential for the patient's upright position, as well as of those which have a synergic function in relation to the paralysed muscles and can compensate for their loss. The most important in this respect, apart from the abdominal muscles, which, because of their insertion points at the pelvis, are essential for restoring walking capability by tilting the pelvis upwards, are the latissimus dorsi, serratus anterior, trapezius, teres major, pectorals and triceps. Details of the technique of compensatory training, in which sling and spring exercises as well as balancing, dressing and walking exercises, have proved invaluable (Fig. 250) have been published (Guttmann, 1945 and 1946)

Readjustment
of vasomotor
control

Over-
development
of trunk
muscles

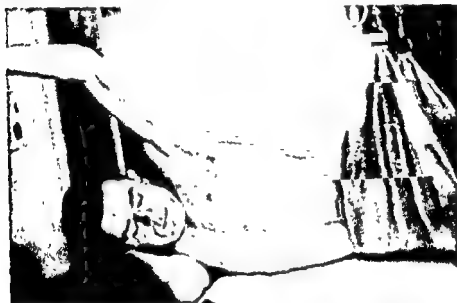
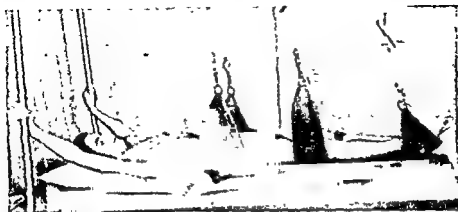


Fig. 251. Swing exercises to promote hypertrophy of the trunk muscles, in a case of complete transverse lesion at T12-T13.

injection of 10–15 cubic centimetres of absolute alcohol made at the interspace between the first and second lumbar vertebrae, may be tried first, as being the less serious undertaking, especially in cases in which the poor general condition of the patient makes a big operation a greater risk (Guttmann, 1947; Gingras, 1948). The immediate effect of such an injection in

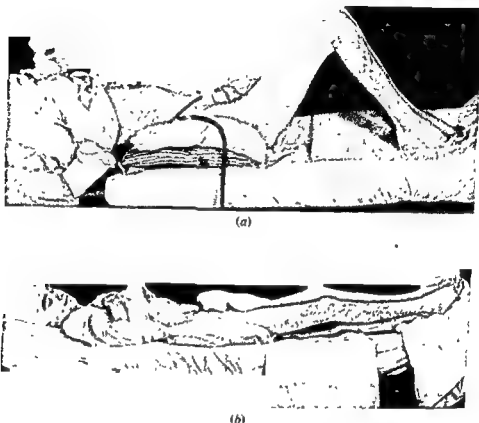


FIG 249.—Flexor spasm (a) before and (b) after intramedullary alcohol injection.

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Readjustment
of vasomotor
control

Over-
development
of trunk
muscles

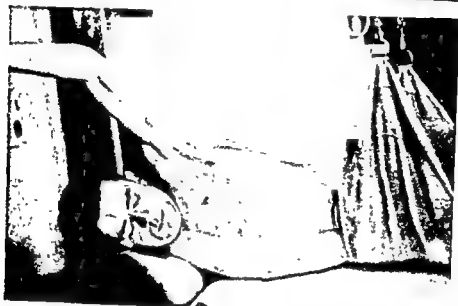
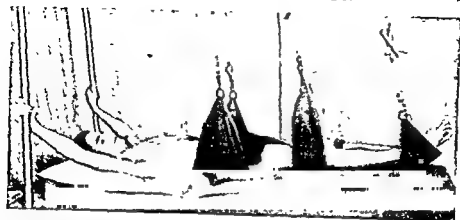


FIG 250.—Sling exercises to promote hypertrophy of the trunk muscles, in a case of complete transverse lesion at Th 1–Th 3.



FIG. 251 —Paraplegics at netball.

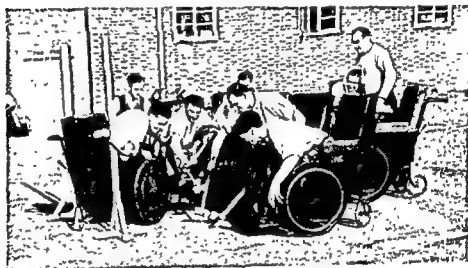


FIG. 252 —Paraplegics at wheel-chair polo



FIG. 253 —Paraplegics at archery.

Games and sports play an important part in the physical and psychological readjustment of paralysed patients. Many games, such as wheel-chair polo, punchball, netball, darts, snooker, skittles and, in particular, archery, can be adapted to the limited abilities of persons with paralysis (Figs. 251, 252 and 253). Experience has clearly shown that a well-trained paralysed person can easily compete with normal persons in a number of games. *Games and sports*

8. SOCIAL READJUSTMENT

Early vocational training in hospital has proved to be an essential step forwards in the social rehabilitation of patients paralysed by spinal cord lesions or brain injuries, as it has restored activity of mind. Rehabilitation by work is started with occupational therapy in the form of simple handicrafts while the patients are confined to bed. It must be remembered that this therapy is not merely occupation as a diversional measure—it is invaluable for the development of the dexterity of the fingers and arms, upon which the future vocation of a paralysed person will depend. As soon as possible, pre-vocational training is added, and experience has shown that many types of training can be successfully employed. *Early vocational training*

In relation to domestic and industrial resettlement, one has to distinguish between two main groups of paralysed persons. The first group will be able and will prefer to return to their homes, if these can be adjusted to their disabilities or if special bungalows can be built for them; it has been proved that these patients are able to return to useful social life, working either at home or in factories and offices. The second group consists of those patients who will not be able to return to their own homes. For these patients, a satisfactory solution is the provision of permanent settlements adapted to their disabilities, in which married and unmarried patients can live with their families in sheltered conditions, either going out in wheel-chairs to their places of employment or doing some form of work inside the settlement. *Domestic and industrial resettlement*

BIBLIOGRAPHY AND REFERENCES

- Abraham, E. P., and Duthie, E. S. (1946). *Lancet*, 1, 455.
 Bell, J. G. Y. (1947). *Proc. R Soc Med*, 40, 441.
 Emmett, J. L. (1943). *J. Urol.*, 49, 815
 — (1947). *Ibid*, 57, 29.
 Gingras, G. (1948). *Treatment Serv. Bull. Canad Veter Adm*, February, 1
 Guttmann, I. (1937). *Klin. Wschr*, 35, 1212
 — (1945). *Med. Times*, N Y, 73, 318.
 — (1946). *Brit. J. phys Med*, 9, 131 and 162
 — (1947). *Proc. R Soc. Med*, 40, 219
 — and Whitteridge, D. (1947). *Brain*, 70, 361
 Head, H., and Riddoch, G. (1917). *Brain*, 40, 188
 Hewitt, W. L. (1947). *Amer J. Med*, 2, 474
 Kremer, M. (1942). *Quart. J exp Physiol*, 31, 337
 Laver, C. H. (1917). *Guy's Hosp Gaz*, 31, 71
 Lawrie, R. S., and Nathan, P. W. (1939). *Lancet*, 2, 1072
 Macalpine, J. II (1948). In *Textbook of Genito-urinary Surgery*, p 356. ed by H P. Winsbury-White Edinburgh, Livingstone
 Munro, D. (1943). *New Engl J Med*, 229, 6.
 Petroff, R. P., and Lucas, F. V. (1946). *Ann Surg.*, 123, 808

- Pulaski, E. J. (1946). *Ann. Surg.*, 124, 392.
Riches, E. W. (1943). *Brit. J. Surg.*, 31, 135.
Shelden, C., and Bors, E. (1948). *J. Neurosurg.*, 5, 385.
Stewart, O. W. (1942) *Lancet*, 1, 287.
Suby, H. I., Suby, R. M., and Albright, F. (1942). *J. Urol.*, 48, 549.
Talbot, H. S. (1946). *Virginia med. Mon.*, 52, 449.
Thompson, G. J. (1945). *Nav. med. Bull., Wash.*, 45, 207.
Wolinsky, E., and Strecken, W. (1946). *Proc. exper. Biol. and Med.*, 62, 162.

[References to other titles are given under Paralysis—Management of, in the Index Volume.]

PARATHYROID GLAND— DISEASES

BY GEOFFREY KEYNES, M.D., F.R.C.S.
EMERITUS SURGEON, ST. BARTHOLOMEW'S HOSPITAL, LONDON

259.] Disease of the parathyroid glands may be manifested either as hyperparathyroidism or as hypoparathyroidism—that is, as an excess or a deficiency of the characteristic hormone of the gland.

PART I HYPOPARATHYROIDISM

This may arise spontaneously by atrophy of the gland; more often it is man- *Causation*
made and results from removal of part or all of the normal parathyroid tissue
during operations on the thyroid gland. It is characterized by symptoms,
due to hypocalcaemia, such as tingling in the extremities, tetany or even
generalized convulsions.

In women the symptoms become worse during the menstrual periods. The
condition is treated by administration of Parathormone, with large doses of *Methods of*
calcium lactate or of calcium gluconate. Often the symptoms tend to subside *treatment*
since any remaining traces of parathyroid tissue hypertrophy and thus adjust
the endocrine balance. Sometimes the symptoms are persistent. Grafting of
heterogeneous parathyroid glands has been tried, but no permanent results
have been obtained, the grafts being absorbed and disappearing within a
short time. The only treatment, therefore, is medical.

PART II HYPERPARATHYROIDISM

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1. INTRODUCTION

This is sometimes regarded as synonymous with generalized osteitis fibrosa
or osteitis fibrosa of von Recklinghausen. It is important, however, to recog-
nize that hyperparathyroidism, with hypercalcaemia and a negative calcium
balance, may be present without there being any recognizable change in the

Association
with renal
calculus

Parathyroid
hypertrophy

Meticulous
examination
necessary

skeleton. Instead, there may be large or multiple renal calculi, causing characteristic renal symptoms, associated with the excessive excretion of calcium in the urine. Routine estimation of serum-calcium and of serum-phosphorus levels in all patients with renal calculi will reveal parathyroid abnormalities in a small proportion of cases. Again, it is sometimes assumed that hyperparathyroidism is necessarily associated with the presence of a parathyroid tumour. Although a tumour is the commonest cause, it is not the invariable cause. Hyperparathyroidism may be due to simple enlargement of normal parathyroid bodies or even to over-action of apparently normal glands. The surgery of hyperparathyroidism is, therefore, directed to the removal of a tumour of the gland if such is present, or to the removal of two or more of the parathyroid bodies if a tumour cannot be identified. Surgical treatment must never be undertaken without the most meticulous examination of the patient by clinical, radiological and biochemical methods.

2. CLINICAL PICTURE

The clinical signs and symptoms of hyperparathyroidism are very varied, and at first are unlikely to be related to the skeleton. It will be easiest to convey a complete picture of the disease by describing briefly the history of a patient who had suffered from the disease for 7 years from the age of 18 before he was operated upon, and who presented every sign and symptom that has yet been recorded (Keynes and Taylor, 1933). These are given in chronological order of appearance.

Characteristic
symptoms

(i) *Initial stage.*—January 1925: The symptoms were acute abdominal pain, persistent vomiting and wasting, at the same time frequency of micturition, due to polyuria, was present. A small calculus was passed *per urethram*. The symptoms persisted with variations in intensity until August 1926, when the patient was found to have a slight albuminuria and gastric atony.

(ii) *Second stage.*—In November 1926 symptoms began to be related to the skeleton. The patient complained of pain and stiffness in the right knee joint. In May 1928 he presented, in addition, a diffuse bony swelling of the right maxilla (Fig. 254), which showed on exploration the structure of an osteoclastoma. A similar swelling appeared in the mandible in February 1930. In July 1931 the patient complained of aching in the bones of the limbs and persistent headaches. Wasting became more pronounced and dyspnoea and severe lassitude were present.

(iii) *Third stage.*—In January 1932 the patient suffered a spontaneous fracture of the right humerus (Fig. 255), and there was pronounced muscular atony. At this time he had brilliantly blue sclerotics also.

At this stage the skeletal changes were fully developed and were as follows: *Generalized fibrocystic disease of the skeleton* was shown radiologically to be most pronounced in the right humerus at its lower end, in the radius on both sides, in the bones of both hands (Fig. 256), and in the long bones of both lower limbs (Figs. 257 and 258). The vault of the skull showed thickening and fluffiness of the bones (Fig. 259), and there was metastatic calcification of the whole of the synovial membrane of the right knee joint and of the insertion of the quadriceps femoris into the patella (Fig. 258).

(iv) *Head and face.*—The forehead was high, broad and square, the whole head being large in proportion to the body. The right malar region was prominent, the mandible was rounded and thickened.

(v) *Thorax.*—The chest was narrow and cylindrical, tapering upwards from the waist. Kyphosis and scoliosis of the dorsal spine and numerous swellings on the ribs were present.

(vi) *Abdomen and pelvis.*—The abdomen was prominent and lax; the pelvis was wide, with prominent crests.

(vii) *Lower limbs.*—The great trochanters were prominent and raised, in association with coxa vara. The femora and tibiae were thickened and curved, with genu varum; there was limitation of movement in the right knee joint.



FIG. 254.—Osteoclastoma of maxilla.



FIG. 255 —Osteitis fibrosa of humerus, with spontaneous fracture

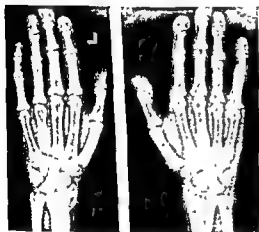


FIG. 256 —Osteitis fibrosa of bones of hands.



FIG. 257.—Osteitis fibrosa of radius and ulna

FIG. 258 —Osteitis fibrosa of femur and tibia, with calcification around the knee joint





FIG. 259.—Osteitis fibrosa of skull.



FIG. 260.—Deformities in osteitis fibrosa.



FIG. 261.—Deformities in osteitis fibrosa

FIG. 262.—Deformity of hands in osteitis fibrosa



In July 1932 a parathyroid tumour was removed from the neck, and all symptoms rapidly abated. Four months later recalcification was apparent in the skeleton, and the bones were ultimately restored to their full strength, so that the patient was able to play football and to work as a golf professional. Nine years after operation he died of uraemia.

3. DIFFERENTIAL DIAGNOSIS

The clinical picture of a classical example of generalized osteitis fibrosa as described above can hardly be mistaken, but confusion occasionally arises in diagnosis when considering patients with lesions in a single bone—that is, with “focal osteitis fibrosa”. There is no evidence to indicate that single lesions, or even those lesions limited to one side of the skeleton, have any connexion with parathyroid disease, and it is, therefore, essential to apply rigidly the biochemical test concerning calcium and phosphorus metabolism before undertaking any operation. Although the normal figure for serum calcium is commonly about 10 milligrams and that for plasma phosphorus about 2.5–3.5 milligrams per 100 cubic centimetres, there may be a normal variation within fairly narrow limits, and estimations of the values should always be undertaken on more than one occasion. In parathyroid disease the serum calcium will rise above 12 up to 17 or 18 milligrams per 100 cubic centimetres, and the phosphorus level may fall below 1 milligram.

Other conditions to be distinguished in the course of differential diagnosis have been mentioned by Hunter in his article on Bones—Metabolic Dystrophies in Vol. 2, p. 279, but in only one—multiple myeloma—is the serum calcium ever raised. In the majority of the patients the presence of Bence-Jones protein will help to differentiate this disease from hyperparathyroidism.

4. SURGICAL TREATMENT

When the diagnosis of hyperparathyroidism has been established, surgical treatment must be undertaken without undue delay, since there is always a progressive deterioration in the patient's condition, and this continues so long as the excessive drain of calcium from the body persists.

Search will already have been made for signs of a parathyroid tumour in the course of the clinical examination, but this examination is likely to have been negative, for the presence of a tumour can seldom be detected from the outside.

The normal parathyroid glands are four in number and lie in close relationship to the posterior border of the thyroid gland. Usually the inferior parathyroid on either side can be identified fairly easily as a small oval long and weighing 30–40 milligrams, which is anterior to the thyroid artery. The superior parathyroid is found similarly related to the superior thyroid arteries. It has been stated that the parathyroids are sometimes contained within the capsule of the thyroid gland, but probably this is untrue

They may, however, be situated in furrows on the surface of the gland, and so appear to be embedded in its substance. Apart from this there are many variations in the disposition of parathyroid tissue. Not infrequently there are more than four parathyroids, as many as seven having been counted, but occasionally they are fewer in number. It is the decrease in number which may be held to account, to some extent, for the incidence of hypoparathyroidism after thyroidectomy.

It is, however, variation in the position of the parathyroids that is more important from the surgical standpoint. The range of this variation is from a position high in the neck to one deep in the thorax. The explanation of this extreme variation is to be found in the embryological origin of the glands. They are derived, according to the most recent investigations (Norris, 1937): (1) from primordia in the fourth branchial cleft, which also give rise to the lobes of the thyroid gland and (2) from primordia in the third branchial cleft in association with the lobes of the thymus gland. It is usually the glands from the fourth cleft which come to lie in the upper position and those from the third cleft which come to lie below, this lower position of the embryologically upper glands being achieved in conjunction with the migration of the thymus gland, the greater part of which is ultimately situated in the anterior mediastinum. This associated migration of thymus gland and lower parathyroids determines the fact, of great importance surgically, that many parathyroids, both normal and abnormal, are situated within the thorax, even as low as the lowest limit of the thymus gland—that is, some way down on the pericardium. The importance of this abnormality is illustrated by the demonstration by Cope in 1941 that of 58 adenomas of the parathyroids identified at the Massachusetts General Hospital, only 42 were in the neck, 11 being in the anterior mediastinum and 5 in the posterior mediastinum. The position of the tumours in the anterior mediastinum may be anywhere from the level of the sternal notch to the lower limit of the thymus gland, and occasionally they are found to be embedded in the thymus gland. Very few of these can be reached through an incision in the neck. On the other hand those in the posterior mediastinum will not have descended so low; not infrequently they will be found lying behind the common carotid artery, and can be reached through an incision in the neck. Sometimes a branch of the inferior thyroid artery affords a clue as to the position of the tumour and can be used as a guide.

Of those tumours situated in the neck, very few can be palpated from without, and even when a supposed tumour has been palpated it may prove on examination to be a nodule of thyroid tissue. Parathyroid tumours are not large—they are usually from 2 to 4 centimetres in diameter—and can never be demonstrated by radiography. Clearly, therefore, any surgeon who sets out to treat hyperparathyroidism by operation must be prepared to search

discovered in that area, he must then be ready to explore the anterior mediastinum, and this exploration may entail the removal of the thymus gland.

In view of these facts the operation may have to be performed in two stages, since it may be inadvisable to explore the mediastinum until material obtained from the dissection of the neck has been fully examined.

Post-operative hypoparathyroidism

Parathyroids situated within the thorax

Tumours situated in the neck

Removal of thymus gland

Two-stage operation

5. THE OPERATION

(1) First stage

The exploration of the neck can be carried out by means of the usual exposure for operations on the thyroid gland, and therefore this need not be described in detail here. The surgeon will first search behind the thyroid gland on either side. If the tumour is not in one of the usual positions of the parathyroid glands, it may be located in close relationship to, or occasionally even behind, the oesophagus. At the same time great care must be taken to identify the normal parathyroid bodies, since, if the hyperparathyroidism is due to diffuse hyperplasia of these instead of to a tumour, one, two or three out of the four bodies should be removed according to their size. Parathyroid tissue estimated to weigh 30 milligrams and over is stated to be enough to prevent tetany and to maintain normal calcium metabolism (Black and Sprague, 1947). Parathyroid bodies should, however, never be removed unless they are definitely hyperplastic, because severe tetany would then follow the subsequent removal of a tumour. *Location of tumour*

The exploration of the neck on each side should be completed by blunt dissection into the upper part of the posterior mediastinum, and by examination of the anterior mediastinum just below the sternal notch.

(2) Second stage

If the exploration of the neck has produced nothing abnormal, then the anterior mediastinum must be exposed. This is best effected by a trans-sternal approach, the sternum being split down the middle as far as the third intercostal space, with lateral cuts into the third spaces so that the two halves of the sternum may be freely raised on either side. A fully controlled anaesthetic of cyclopropane and oxygen is administered. *Trans-sternal approach*

The steps of the operation are as follows.

(a) The superficial tissues above the sternal notch and over the centre of the sternum are infiltrated with 1 in 500,000 adrenaline hydrochloride in normal saline, in order to diminish bleeding from small vessels. This is not essential.

(b) An incision of about 2 inches is made through the skin and platysma muscle in the line of the previous collar incision for exploration of the neck. Flaps are dissected up and down for a short distance. Another incision at right angles to the first is made downwards over the centre of the sternum as far as the upper border of the fourth rib. This is carried down to the bone. *Incision*

(c) The pretracheal muscles are separated for a short distance above the sternal notch and an index finger is introduced behind the sternum as far as it will reach, in order to separate connective tissue and muscular attachments from the back of the bone. The upper part of the pleura is also pushed away by this manoeuvre.

(d) The third intercostal space is defined on each side by reflecting muscles and periosteum with a rugine. A blunt dissector is introduced upwards through each intercostal space and made to meet a finger introduced from above. In this way the pleura is pushed aside for the whole distance so that it will not be injured in the next step.

(e) The sternum is split down the centre as far as the third space with Sauerbruch's sternum-splitter or Lebsche's chisel, or, if these instruments are not

available, with a small saw, a copper strip being introduced beneath the bone to protect the contents of the mediastinum. A lateral cut from the lower end of the split outwards into either intercostal space releases the two halves.

(f) The two halves of the sternum are raised and held apart with a strong self-retaining retractor.

*Exposure of
thymus
gland*

(g) If the tumour is not seen at this stage in the anterior mediastinum, the thymus gland is carefully exposed by blunt dissection, the pleura being stripped away from the gland without injury. If necessary, the thymus gland is removed.

Sutures

(h) The mediastinum is closed by suturing together the two halves of the sternum by means of strong catgut passing through muscles and periosteum on the surface of the bone. It is unnecessary to wire the bone, which quickly unites. The catgut knots are buried by suturing the subcutaneous fat over them with fine catgut. The anaesthetist inflates the lungs in order to expel blood from the mediastinum, which is not drained, and the skin incision is closed.

6. POST-OPERATIVE TREATMENT

If a parathyroid tumour has been found and removed, the serum-calcium concentration will fall very rapidly, and it is common for the patient to show signs of parathyroid deficiency within a few hours of the operation. The first symptom will be tingling in the fingers and toes and this may not develop into actual tetany. Precautions should be taken, however, to forestall the occurrence of tetany, and this may be achieved by giving large doses, up to 300 grains, of calcium lactate or of calcium gluconate by the mouth at short intervals. It is usually unnecessary to give any parathyroid hormone preparation although, if the symptoms are severe, this method of treatment will accelerate recovery. Within a few days the activity of the normal parathyroid tissue reasserts itself and the tendency to tetany disappears.

Tetany

*Estimation
of serum-
calcium level*

The effect of the operation will be observed by means of daily estimations of the serum-calcium level, beginning on the first day of the post-operative period. It will usually be found that the serum-calcium level has fallen quickly to well below normal, that is, to 7-9 milligrams per 100 cubic centimetres, and that it will rise again to normal in the course of a few days. The initial fall will prove the success of the operation, but this will, of course, be verified by histological examination of the extirpated tumour. The tumour may be solid or partly cystic, but in either case the microscopic appearance is that of parathyroid tissue.

7. RESULTS OF OPERATION: PROGNOSIS

The symptoms of hyperparathyroidism begin to disappear soon after removal of the tumour, but it will be a long time before any change will be perceptible in the radiographic appearance of the bones. Recalcification, however, will gradually take place, and the administration of extra calcium by the mouth, in the form of calcium lactate, may assist the process. Ultimately, in the course of two or three years, the bones will become radiologically as dense as normal, though still showing irregularities of structure (Figs. 263 and 264), and all tendency to pathological fractures will be abolished although the deformities will remain. The kidneys may contain small undetected calculi, and renal

*Recalcification
process in
bones*



FIG. 263.—Left radius and ulna 6 years after removal of parathyroid tumour.



FIG. 264.—Right radius and ulna, 6 years after removal of parathyroid tumour, with radius fractured by ordinary degree of violence

or ureteric colic is apt to occur, perhaps with impaction of a calculus in the ureter, so that secondary operations may become necessary. Apart from this, the ultimate prognosis should be guarded, particularly if the disease was of long duration before the tumour was removed. It may be that serious injury has been caused to the kidney by deposition of calcium, and the patient may die of renal insufficiency within ten or twelve years, but, although a fairly large number of examples of parathyroid tumour have been described in the literature since the first case recorded by Mandl in 1926, there have not been many reports published of the results after ten or more years.

REFERENCES

Black, R. M., and Smith, R. C. (1937) *Brit. J. Surg.*, 22, 73

C

Mandl, F. (1926) *Arch. klin. Chir.*, 143, 1.

Norris, E. H. (1937) *Publ. Carneg. Inst.* No. 479, p. 247

[References to other titles are given under Parathyroid Gland—Diseases, in the Index Volume. The subject is also dealt with in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 424.]

PELLAGRA

By HUGH S. STANNUS, PH.D., M.D., F.R.C.P., D.T.M. & H.
CONSULTING PHYSICIAN, FRENCH HOSPITAL, LONDON

260.] Pellagra on first consideration would appear to have no place in a work on surgical practice. Whereas it is true that there are no complications in this disease calling for surgical intervention, pellagra may supervene in many conditions within the province of the surgeon.

Gastro-intestinal conditions

These conditions are nearly all associated with diseases of the gastro-intestinal tract either directly—especially if diarrhoea or vomiting has been present—or more indirectly on account of a restricted or unbalanced diet either before or after operative intervention. Pellagra occurring in these circumstances was at one time considered to be different from ordinary endemic pellagra and was called “secondary” pellagra, but it was realized some years ago that the diseases were one and the same. Hence it is inadvisable to use the term “secondary” pellagra.

Susceptibility in latent deficiency states

Apart from these diseases of the gastro-intestinal tract it must never be forgotten that, in any subject suffering from a latent deficiency state, operation or even the administration of an anaesthetic or the giving of a purge may precipitate an “attack” of pellagra, the first symptom being an unspecific intractable diarrhoea.

Association with lymphopathia venerea

Among those conditions with which pellagra has been associated more commonly, the following may be mentioned: strictures, ulcers and new growths of any part of the gastro-intestinal tract; after gastrectomy, short-circuiting operations and excisions; tuberculous and ulcerative colitis, diaphragmatic hernia, cirrhosis of the liver, cholelithiasis and cancer of the pancreas. Lastly it may be associated with the paraproctitis and rectal stricture due to infection with the virus of lymphopathia venerea (lymphogranuloma inguinale when the inguinal glands are affected), which is more common in the female and is sometimes complicated by recto-vesical or recto-vaginal fistula. Pellagra is not uncommon in areas where lymphogranuloma prevails.

The relationship between pellagra and diseases of the large bowel is, of course, particularly interesting in view of the fact that the biosynthesis of many vitamins in the colon may prove of importance.

It is unnecessary here to give any full description of the symptoms of pellagra. Though the so-called classical symptoms of the text-book may be seen, it is of much greater importance to recognize those lesser signs which should lead the surgeon to review the patient's dietary.

Diagnostic signs

These signs may be extremely varied. A change in the patient's mental state is possible—he may become depressed, morose or apathetic or he may appear to be suffering from neurasthenia. Again, he may exhibit a mild psychosis—complaining of insomnia, giddiness, disturbances of hearing and vision, paraesthesiae or burning pains in the feet. Examination may reveal an acute glossitis, a stomatitis involving also the soft palate and pharynx associated with dysphagia, cheilosis and an angular stomatitis with a similar condition about the nares, prepuce, anus or vulva together with a scrotal dermatitis; a

history of unexplained watery diarrhoea may be elicited. The symptoms may precede the appearance of a dull brown-red erythema on the backs of the hands with, perhaps, a violaceous thickening of the skin over the interphalangeal joints, though in the patient confined to bed there may be no more than dull pigmented areas of thickened skin over pressure points on the limbs.

Although it is to some extent possible to ascribe certain of the symptoms of pellagra to deficiencies of specific vitamins, the disease should be regarded preferably as one due to a deficiency in varying proportions of the B₂ group of vitamins plus a deficiency of protein, to which may be added a deficiency of vitamin B₁.

Treatment will consist in ensuring a full and balanced diet which the patient can absorb, including milk, eggs, meat and so forth, in conjunction with yeast, liver extract, plasma and similar substances, supplemented, if necessary, by vitamins.

[References to other titles are given under Pellagra in the Index Volume. The subject of Pellagra is also dealt with in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 468.]

PELVIC ORGANS—DISPLACEMENT

BY SIR WILLIAM FLETCHER SHAW,
M.D., F.R.C.P., F.R.C.O.G., M.M.S.A. (Hon.), F.A.C.S. (Hon.)
EMERITUS PROFESSOR OF OBSTETRICS AND GYNAECOLOGY, MANCHESTER
UNIVERSITY; CONSULTANT SURGEON FOR WOMEN, ST. MARY'S HOSPITAL,
MANCHESTER; CONSULTANT GYNAECOLOGIST, MANCHESTER ROYAL INFIRMARY

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1. DEFINITION

261.] Many displacements of the female pelvic organs are described, but only three of these call for operative treatment—retroflexion of the uterus, prolapse of the ovaries and genital prolapse.

2. RETROFLEXION OF THE UTERUS

(1) History

This, during the latter half of the last century, was one of the most abused conditions in gynaecology. It was easily recognized, and for a long time it was considered to be the cause of almost any pelvic symptom and of many general symptoms.

Not only was the condition easily recognized but, provided that the uterus was not fixed by adhesions, it could be replaced and held in its new position by one of the numerous pessaries which the gynaecologists of the last century devised. It was also the reason why so many women of the Victorian period were condemned to a life of semi-invalidism on a couch or restricted to the gentlest of exercise. At the turn of the century, as the abdomen was more frequently opened, operation after operation was devised to fix these unfortunate organs into a position which the majority had not previously occupied and, as these operations were simple to perform compared with many other abdominal operations, countless numbers of women were condemned to undergo them unnecessarily. Naturally, after such enthusiasm came reaction, and some gynaecologists went so far as to say that retroflexion of the uterus never produced symptoms and never called for treatment.

Now we can take a more balanced view and recognize that the vast majority of women with a retroflexed uterus do not suffer any symptoms from this condition, but a small percentage do, and they are relieved when the uterus is kept in an anteфлекed position.

(2) Aetiology

Retroflexion is a very common position of the uterus both in nulliparous and in parous women. In the former the uterus has developed in this position, but in parous women the retroflexion may be a return to normal, or it may have acquired this new position during the puerperium.

(3) Symptoms

There are two symptoms which may be due to retroflexion of the uterus—chronic aching pain in the back, and sterility. It cannot, however, be emphasized too strongly that these symptoms may be produced by many other conditions and, therefore, that an operation must not be advised for this condition until it is tolerably certain that it is the retroflexion which is the cause of the patient's symptoms.

(a) *Backache*

Two of the common causes of chronic pain in the back in parous women are strain of the sacro-iliac joints and early genital prolapse. These must be borne in mind when making a diagnosis. *Chronic pain in the back*

There are also two conditions commonly associated with retroflexion which are more likely to be the cause of the pain than the actual position of the uterus—pelvic adhesions and prolapse of the ovary. It is necessary, therefore, to examine carefully for both of these conditions

If the retroflexed uterus is fixed by chronic adhesions relief of the pain in the back is often obtained by tampons of glycerin and Ichthyol. One should be inserted well into the vaginal fornices, removed 24 hours later, a hot douche of boric lotion (1 drachm to the pint) given and another tampon inserted. The treatment should be continued for at least a month except during menstruation. *Adhesions*

If this has been tried and has failed the only treatment is an exploratory laparotomy, in the hope that the adhesions can be freed and that the uterus can be retained with at least one healthy ovary and tube. It is, however,

always advisable to obtain permission beforehand to do a radical operation in the event of the adhesions being found to be so dense that it is impossible to retain any healthy organs.

When the adhesions are freed and it is decided to retain the uterus, the organ is lifted up and retained in this position by Gilliam's operation.

If a retroflexed uterus which is causing backache is mobile, it should be replaced and retained in position by a Hodge pessary. If this cures the pain, and if the patient objects to the continued use of a pessary, an operation to fix the uterus forward can be performed.

Prolapse of one or of both ovaries frequently accompanies retroflexion of the uterus. In these cases it is the prolapsed ovary which produces the pain rather than the retroflexed uterus. This is dealt with on page 481.

(b) Sterility

Infertility also may be produced by many conditions, and it is only after careful elimination of these in both the husband and wife that the blame may be put upon the retroflexion of the uterus if this is found to be present. Retroflexion of the uterus is not an absolute barrier to pregnancy, but it is a relative one because the retroversion which generally accompanies the retroflexion points the cervix forward into the anterior fornix where, in many instances, it is elevated above the pool of semen deposited in the posterior fornix. In some instances impregnation can be procured by coitus in the reversed position, or by the replacement of the uterus to the normal position, followed by the insertion of a Hodge pessary.

If the patient objects to wearing a pessary the uterus may be held forward by an abdominal operation. This replacement of the uterus into a normal position which replaces the cervix into the posterior fornix may be followed by pregnancy, provided that care is taken beforehand to eliminate any other cause of sterility in both partners.

(4) Operative treatment

Operations to fix the uterus forward are legion. Probably the most popular is Gilliam's operation, or one of its numerous modifications.

The peritoneal cavity is opened through a median longitudinal or Pfannenstiel incision. A loop of ligature material is attached to each round ligament about 1 inch from the uterine end and held by forceps. The fascia covering the rectus muscle is incised in a longitudinal direction about half an inch from the middle line and $1\frac{1}{2}$ inches above the symphysis pubis and a hole is made about half an inch in length. Through this hole a pair of slightly curved forceps are passed, superficial to the rectus muscle and then between the fascia and peritoneum until the internal abdominal ring is reached. The point of the forceps is then directed along the course of the round ligament, beneath the peritoneum covering the latter, until the ligature on it is almost reached. The peritoneum is opened upon the point of the forceps and the free end of the ligature grasped in the forceps which are withdrawn bringing the ligature out of the hole in the rectus sheath. Sufficient steady pressure is made upon the

the incision in the fascia and on the other side. The abdo-

minal incision is closed in the usual way.

Mobile
retroflexion

Prolapsed
ovary

Sterility

Gilliam's
operation

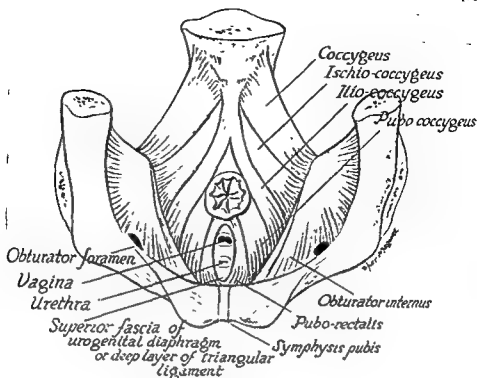


FIG. 265.—Diagrammatic drawing of the muscles of the pelvic floor, viewed from above (After Maguire)

muscle. Below this is another layer of muscle and connective tissue, the triangular ligament, attached to the pubic rami on each side. This supports the

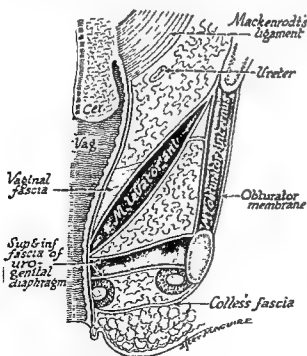


FIG. 266.—Diagrammatic drawing of parametrium, levator ani and triangular ligament (After Maguire)

lower part of the vagina and supplies the sphincter action of the urethra which runs obliquely through it. In the midline this muscular floor is perforated by three tubes, the urethra, vagina and rectum, and in each case muscle fibres are carried downwards surrounding and attached to the tube (Fig. 266).

Another important structure, the parametrium, enters into the formation of the pelvic floor. This consists of unstriated muscle fibres and connective tissue, and is collected chiefly in the concavity of the pelvic muscular floor in much the same way as liquid cement would collect

if poured into it. The main part collects around the cervix and the uterine vessels, with prolongations forwards into the utero-vesical ligaments and backwards into the utero-sacral ligaments. Mackenrodt's ligament is the upper and firmer portion of the parametrium.

It is the pelvic floor, this combination of muscle and parametrium, inserted into the cervix and upper part of the vagina, which suspends the uterus in its mid-pelvic position, and combined with the perineal musculature preserves the vaginal walls. This is easily demonstrated when performing a total hysterectomy. When the uterus is grasped and pulled up from the pelvis, section of the broad ligament and other ligaments has no effect, but as soon as the cervical attachments are cut up comes the uterus with a jerk.

(3) Aetiology

A firm, well-developed, healthy pelvic floor is essential to the maintenance of the uterus and vaginal walls in their normal position, and it is only when this pelvic floor is damaged or poorly developed that any form of genital prolapse can occur.

In the great majority of patients with genital prolapse the laxity of the pelvic floor is brought about by damage during parturition. A small percentage of these patients, however, are nulliparae in whom poor development of the pelvic floor is the chief aetiological factor, to which has been superadded one of the secondary causes of prolapse. *Cause of weakness of pelvic floor*

These secondary causes play an important part in both multiparae and nulliparae and, although they will not be able to produce genital prolapse in the absence of a weakened pelvic floor, they often increase the extent of the prolapse or determine its onset when the pelvic floor is only slightly damaged or weakened. *Secondary causes*

These secondary causes are: (1) Increased abdominal pressure produced by hard work and chronic cough. (2) Conditions pushing down the pelvic contents, such as a heavy uterus, fibroids, subinvolution and abdominal tumours (3) Conditions dragging down the cervix, such as a heavy cervix due to chronic cervicitis or fibroids. A heavy uterus due to subinvolution, or a heavy cervix due to chronic cervicitis is commonly found in multiparae and, therefore, these conditions are often associated with genital prolapse the onset of which they have hastened. Increased abdominal pressure comes into play in old women with chronic bronchitis, and frequently it is the determining factor in producing genital prolapse in women who have sustained damage to the pelvic floor at a confinement many years before. *Heavy uterus and cervix*

Heavy physical work is the cause of genital prolapse in virgins and in nulliparae with weakly developed pelvic floors. Some years ago Lancashire was one of the few centres where this condition was commonly found. Now, with the long years of depression in the cotton trade, these cases are not so frequent, whereas they have appeared in London since that area became more industrialized. *Coughing*

(4) Symptoms

Most patients with genital prolapse complain of "something coming down", though in the early stages the main complaint may be an aching pain in the lower back and abdomen which is made worse by standing or by hard work. *Heavy work*

In a small minority of cases the patient complains of stress incontinence of urine. These patients are dry during the night and most of the day, but void a small quantity of urine when they bend down or strain.

Parity

The great majority of patients with genital prolapse are parous and more commonly multiparous women, and most of them date the commencement of their symptoms from a confinement; if this is not the last one they usually state that the symptoms have increased with each successive labour. Some parous women, however, date the commencement of their symptoms to a time many years after the last confinement. In these cases the pelvic floor was damaged at the confinement, but the descent of the tissues was slight and it was only following atrophy of the pelvic muscles after the menopause, or following the onset of one of the secondary causes of prolapse such as chronic cough or a pelvic tumour, that the patient became conscious of "something coming down". In a small percentage of cases the patient is nulliparous or *virgo intacta*. In these patients there is some developmental weakness of the pelvic floor and almost always there is a history of increased abdominal pressure, usually from heavy work.

Nulliparae

(5) Physical signs

On inspection of the parts it is seen that the vaginal tissues bulge downwards when the patient strains. Sometimes the whole uterus comes outside the vulva—*procidentia*; or the cervix descends without complete extrusion of the vaginal walls—*prolapsus uteri*; or the chief bulge is seen to be the anterior vaginal wall—*cystocele*; or the posterior wall—*rectocele*; or the straining may be accompanied by a spurt of urine—*stress incontinence*.

Degree of prolapse

Any one of these may be the main feature, but it is very rarely that laxity of one part is found without a similar condition being present in a lesser degree in other parts. In *prolapsus uteri* it is more common to find the uterus in the retroflexed position, though this is not an invariable feature.

In early cases (the type in which the patient complains of aching pain in the lower abdomen accompanied or unaccompanied by backache after standing or working) there is little bulging to be seen when the patient strains, but two fingers inserted into the vagina find this to be lax and the cervix to be unduly mobile when the patient strains or coughs.

(6) Treatment

This may be either by the wearing of a pessary or by operation.

The pessary.—Only two types of patient can justifiably be subjected to this treatment, because it condemns them to wear a pessary perpetually with the necessary cleansing douches and the periodical medical visits for replacement. Genital prolapse never improves but steadily increases, especially after the menopause, and many patients who have been reasonably comfortable with a pessary in middle life become so miserable with advancing years that they then insist upon something permanent being done. This accounts for the numbers who are operated upon over the age of 60 years.

Young parous woman

If, however, the patient is young and has had only one child, or a small number of children, and intends to have more, it is advisable to allay her

upon an operation, although they have been warned that surgery

may again weaken the tissues and necessitate a second operation. In a series of patients who had confinements after the operation of colporrhaphy, only one-sixth had any sign of recurrence, and in only a very small number was this sufficiently severe to warrant a second operation (Shaw, 1934).

In minor cases a Hodge pessary will sufficiently stretch the vaginal walls, but a more generally useful pessary is one of the round, rubber-covered, watch-spring type. *Type of pessary*

The other group consists of those patients who, from retrogression due to age or concurrent disease, cannot stand the strain of an operation. Age itself is no barrier, only the retrogression which accompanies it, and this varies greatly in the individual patient. Frequently we operate upon patients over 70 years of age, and although I have not seen a patient over 80 years of age upon whom operation was justifiable, one of my colleagues operated upon a woman aged 82 years who lived a number of years to bless him for the relief he had afforded her. *Women unfit for operation*

Rubber-covered watch-spring pessaries are the most generally useful, but in some old patients the musculature is so weak that the skin folds itself over the pessary and stretches down to the vulva. In these a cup-and-stem pessary is often successful.

(7) Operative treatment

Many operations are recommended and can be classified under the following four headings in the inverse order of their usefulness.

(a) Hysterectomy

Vaginal hysterectomy is described in association with colporrhaphy, and under various names, as a cure of prolapse. This it never is.

If a patient with genital prolapse has a pathological condition of the uterus which calls for its removal, vaginal hysterectomy can easily be combined with colporrhaphy. The hysterectomy, however, has no part in the cure of the prolapse; in fact, the colporrhaphy must be stitched more carefully, because recurrent prolapse after hysterectomy is much more difficult to cure than when the uterus is still *in situ*.

It is wrong, therefore, to speak of operations which include hysterectomy as one operation for the cure of prolapse: they are in truth a combination of two operations, a hysterectomy to remove a diseased uterus and a colporrhaphy to cure the prolapse. As a combined operation it is very useful, but only when there is some pathological reason for the removal of the uterus.

(b) Interposition operation

In this operation an anterior colporrhaphy is performed, the bladder dissected from the cervix, the utero-vesical pouch opened and the fundus uteri pulled down into an exaggerated anteflexed position and anchored below the bladder. It is unjustifiable to do this unless the patient, at the same time, is sterilized or has already passed the menopause. I do not describe the operation in detail because I have never seen a case in which it was necessary or justifiable.

(c) Le Fort's operation

In an old patient with such poor pelvic tissues that she cannot retain a pessary and such poor general condition that there is some risk in doing a full

colporrhaphy, this simple operation may be done. A vertical strip of vaginal skin about half an inch wide is dissected from the midline of the anterior vaginal wall from the cervix to near the urethral orifice. A similar strip is dissected from the posterior wall from the cervix to the vulval outlet and these two raw strips are stitched into apposition from the cervix downwards. This produces a permanent median raphe, dividing the vagina into two tubes and on the raphe is permanently perched the uterus. The operation is rarely required.

(d) Colporrhaphy

Manchester operation

This operation will cure practically every type of genital prolapse, early or complete, in the young or old, parous or virgins. By the Manchester operation is meant a combined anterior and posterior colporrhaphy with amputation of the cervix and with special stress on deep suturing of the pelvic floor both before and behind the cervix. Most operators develop minor modifications of technique, but so long as the operation conforms to the description given above it is a Manchester operation, and it only makes for confusion to attach various names to these modifications.

Pre-operative treatment.—These extensive pelvic operations may produce shock, especially in elderly women. The patient, therefore, should be admitted to hospital 48 hours before the operation and should be kept quietly in bed. This allows her to become used to being in bed, and a careful examination can be made for complications which increase the risk of the operation. The bowels should be opened with a gentle laxative the morning before the operation, and on the morning of the operation the lower bowel should be washed out with an enema.

Cervical ulceration

Procidentia is often accompanied by ulceration of the cervix. These ulcers are usually infected, and no matter how carefully they are cleaned before and covered during the operation, there is a great risk of the wound becoming infected. They heal in a short time with proper treatment, and since prolapse does not call urgently for operation it is inadvisable to operate until the ulcer is healed.

The ulceration is due to constriction of the blood supply when the uterus is down, combined with friction from the thighs, and the treatment is removal of the causes. Keep the patient in bed; replace the cervix whenever it comes down but do not insert a pessary; douche twice daily, once with a mild antiseptic and once with alum as an astringent. It is rarely that an ulcer will not heal in a fortnight.

Leucorrhoea

Many cases of genital prolapse have a laceration of the cervix and chronic cervicitis, and with this there is generally a fairly copious discharge. It is, however, rare for the cervix to be heavily infected. If infection is suspected the discharge should be examined and appropriate treatment should be given. A vaginal douche to wash away the discharge is given the morning and night before the operation, nothing further is required beyond the usual cleansing when the patient is on the operating table.

Cystitis

Care must be taken to examine the urine for evidence of cystitis, which is not uncommon in this type of patient. If cystitis is found operation must be deferred until this condition is cured, because the operation interferes so much with the bladder that an acute attack of cystitis is likely to follow, and in some instances the infection reaches the kidney.

(c) *Technique of the Manchester operation*

The patient is placed in the lithotomy position and the operation site is shaved if this has not been done in the ward. The skin of the operation site and the vagina and cervix are carefully wiped dry and painted with iodine. The patient's legs, body and the operating table are covered with sterile sheets. The simplest method is to use one large sheet which covers this whole region, with a triangular opening for the operation site and short wide projections into which the knees and feet of the patient project (Fig. 267)

The cervix is grasped with a pair of vulsellum forceps, dilated, and the uterus curetted. It is advisable to curette all cases; it only takes a few moments and occasionally some pathological condition is discovered.

The description of the operation can be followed clearly by reference to the illustrations.

Fig. 268 shows the prolapsed cervix. The left labium minus has been stitched back over the surrounding sterile towel. When the right labium minus is similarly treated a good view of the vagina is obtained, with a smooth, easily cleansed surface.

Fig. 269 shows the cervix grasped by vulsellum forceps and a triangular area marked out on the anterior wall, with the apex below the urethral orifice and the base above the cervix. This triangle is widest a little distance above the base. In complete procidentia this is an easy procedure, but with the more common case, in which the cervix comes only to the vulva, only the lower part of the triangle can be mapped out at first.

Fig. 270 shows the dissection of the triangular area. In cases of partial prolapse this dissection from the base is especially useful. The dotted line indicates the incision through a few muscular fibres and connective tissue which is necessary to free the bladder.

Fig. 271. The bladder has been separated from the cervix and has been pushed upwards. This exposes the cervix with the parametrium on each side. Two deep sutures are inserted next, which include on each side the anterior fibres of the pubococcygeus muscle and a portion of the parametrium.

When these are tied at a later stage, as shown in Fig. 277, a reef is taken in of the tissues, which are shortened and tied in a thickened mass in front of the cervix. This shortens and thickens the only supports of the uterus and at the same time throws the cervix farther backwards, which in its turn helps to keep the uterus in the position of ante flexion. This is the most important part of the operation, but these sutures must not be tied until the cervix is replaced in the position in which it must be retained.

Fig. 272 shows the method of freeing the cervix. An incision is carried around the posterior part of the cervix joining the angles of the anterior colporrhaphy.

Amputation of the cervix is usually necessary. Not only is the cervix hypertrophied in most cases, but the tying of the sutures shown in Fig. 271 tends further to obtrude the cervix.

Fig. 273 shows the amputation of the cervix.

Figs. 274 and 275 show the method of suturing the vaginal flap over the cut surface of the cervix, the angles of the anterior colporrhaphy being sutured together in the centre of the cervix.

Fig. 276 The cervix is sutured and two or three sutures have been inserted through the anterior vaginal wall and the deep muscle, and so is commenced the suturing

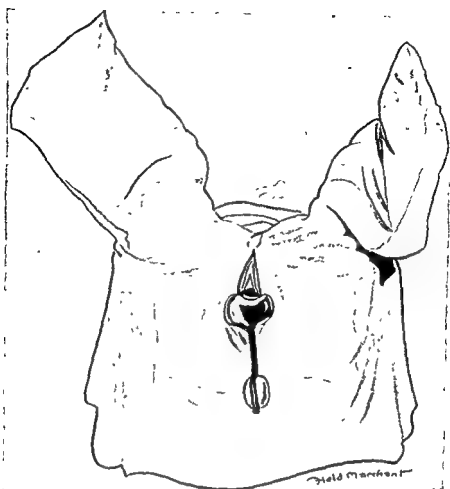


FIG. 267 —One single sheet with wide short pockets for the legs and an opening for the site of operation

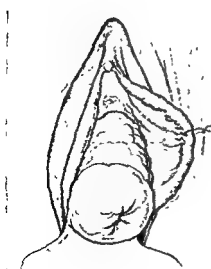


FIG. 268.

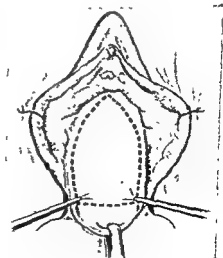
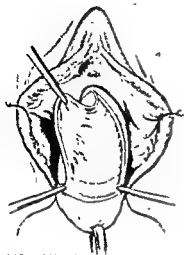
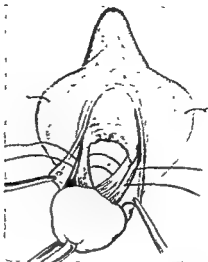
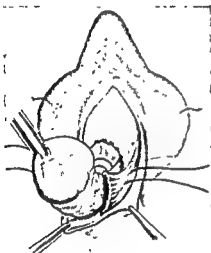
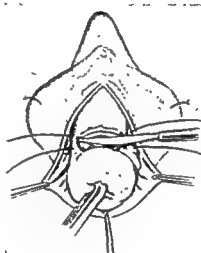
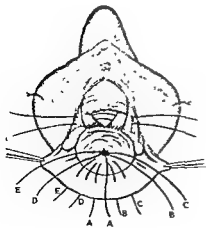
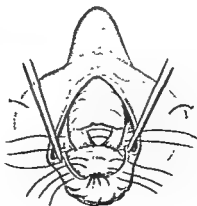
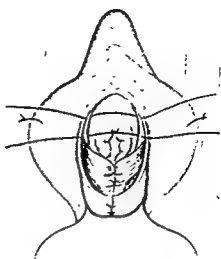
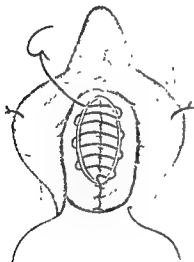
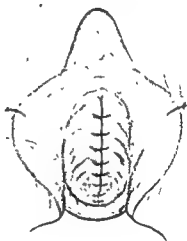
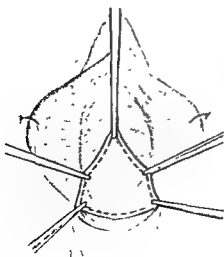
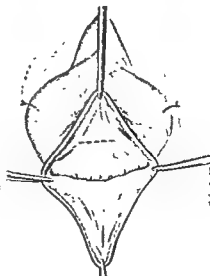


FIG 269

MANCHESTER OPERATION—*cont.*FIG.
270.FIG.
271.FIG.
272.FIG.
273.FIG.
274.FIG.
275.

MANCHESTER OPERATION—*cont*FIG.
276FIG.
277.FIG
278FIG
279FIG
280FIG.
281.

MANCHESTER OPERATION—*cont.*

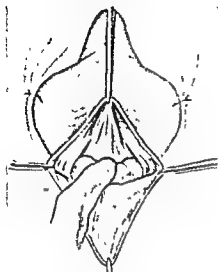


FIG
282

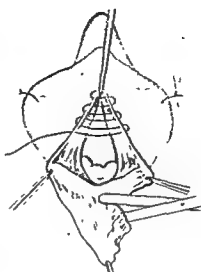


FIG.
283.

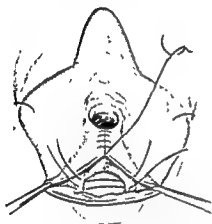


FIG
284

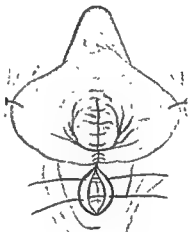


FIG.
285.

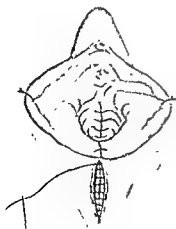


FIG
286.



Distal
segment—

FIG.
287

of the anterior colporrhaphy. The deep sutures inserted in Fig. 271 are still shown untied

Fig. 277. The cervix has been replaced and the uterus put into the position of antelexion and the deep sutures have been tied. If a cervical suture has been left uncut, tension on this will prove that with reasonable traction the cervix cannot then be dragged down.

Further sutures are now inserted in the pubo-coccygeus muscle, the parametrium and the triangular ligament on each side to close the hernia through which the bladder and urethra protruded in a case of cystocele and stress incontinence. The more the bladder and urethra protrude the more carefully must they be freed from adjacent structures and muscle sutured over them. In a case of stress incontinence, after the urethra is freed on both sides, this should be stretched by pushing up the base of the bladder with some blunt instrument and held in this position by an assistant while the muscle is sutured over it.

Fig. 278. This shows the deep sutures in position and a running suture to draw the cut edges of the vagina together. This superficial suture can be either a continuous one, as shown in this diagram, or interrupted. Not infrequently there is some troublesome haemorrhage below the urethra. The simplest way to arrest this is by a mattress suture.

Fig. 279. This shows the anterior colporrhaphy completed.

Fig. 280 shows the commencement of the posterior colporrhaphy. A pair of Spencer Wells forceps grasps the loose tissue near the cervix. Another pair grasps the redundant tissue on each side. The triangular area thus marked out is dissected away and the incision continued to the perineum where the angles are again held with forceps. The amount of tissue removed depends upon the extent of the rectocele and varies in different cases.

Fig. 281 shows the triangular area dissected away and a dotted line indicates the position of the incision to separate the rectum from the vagina.

Fig. 282 shows this separation completed, and indicates some tissue which is part of the levator ani and parametrium at the base of the utero-sacral ligaments.

Fig. 283 shows a running suture inserted through the cut edges of the vagina.

Fig. 284 shows the running stitch drawn tight and the two deep sutures inserted through the levator ani and parametrium. These deep sutures are the most important ones in the posterior colporrhaphy, as they are in the anterior colporrhaphy.

Fig. 285 shows the suturing of the vaginal edges completed. The deep sutures shown in the previous diagram are tied and another pair inserted into another layer of muscle.

Fig. 286 shows the skin of the perineum drawn together with a running suture.

Fig. 287 shows the reconstructed vulva.

Throughout the operation catgut alone is used as a suturing material. For the deep tissues separate sutures should be used, but for drawing together the cut edges of the vagina either continuous or separate stitches do equally well.

At the end of the operation a little gauze soaked in bismuth, iodoform and paraffin or other mild antiseptic emollient should be packed into the vagina. This prevents the gauze from adhering to and damaging the vaginal tissues.

The majority of patients with some form of genital prolapse require the whole operation, though the amount removed from the anterior or posterior walls of the vagina or from the cervix varies in each case. In a small number

only an anterior or only a posterior colporthaphy is necessary and a healthy unenlarged cervix does not require to be removed. Only experience can decide how much must be removed in each case.

(f) *Post-operative care*

The patient will require morphine during the first 24 or 48 hours, but rarely after that period. The vaginal gauze must be removed the morning after the operation. It slips out quite easily, and the majority of the patients then pass urine. The perineum should have the sterile pad replaced whenever it becomes sodden with discharge, and the wound and the surrounding skin should be lightly dabbed with spirit after the patient has passed urine or faeces. An aperient should be given on the second evening; liquid paraffin with cascara is the best. Nothing further is required except to make the patient comfortable. Much harm can be done by over-care of the perineum and by fussiness.

(g) *Complications*

Two common and troublesome complications must be watched for.

(1) *Retention of urine*.—This is a very common complication and some opera- *Retention of urine*
tors now leave a few ounces of mercurochrome, $\frac{1}{2}$ per cent, in the bladder. This produces a mild irritation and causes the patient to void urine herself. If the patient has not passed urine the morning after the operation and does not do so when the vaginal gauze is removed, every encouragement should be given; the patient should be even allowed to sit upright on the bed-pan. If she does not pass urine voluntarily, a soft rubber catheter must be passed into the bladder with all aseptic precautions because an over-distended bladder delays voluntary evacuation. If a catheter is passed on the second morning it is usually necessary to repeat this until the patient's bowels are moved on the third or fourth day, and it is better to repeat the catheterization twice daily to avoid distension of the bladder. The majority of these patients pass urine voluntarily when the bowels are moved, but occasionally it is necessary to continue with the catheter for a few more days. Every effort should be made to encourage the patient to pass urine herself, because every passage of the catheter carries a potential risk of infection. A careful watch must, therefore, be kept on the urine, and any sign of infection calls for treatment of the cystitis. Some operators prefer to insert a self-retaining catheter in all cases, but in my experience this is followed by cystitis even more frequently than is the repeated passage of a catheter.

(2) *Sepsis and haemorrhage*.—The cervix and vagina are potential septic areas *Haemorrhage*
and, no matter how carefully the skin is treated, there is a possibility that part of the incision may not be healed when the catgut sutures give way about the end of the first week and haemorrhage may occur. The bleeding area is generally on the cervix although it may occur in any part of any of the scars. It behoves the nurse, therefore, to keep a careful watch on the pad about this period, and to report the first sign of pink discharge. If it is reported in this early stage it is rarely that anything is required beyond an antiseptic douche through a rubber catheter. This must be passed gently to the top of the vagina so that the douche washes out any septic clot retained near the cervix, and it should be repeated daily for three or four days. Occasionally the haemorrhage is more severe and does not stop with douching, though this is rare if the douche has been given on the first appearance of the pink discharge.

In these more severe cases the vagina should be packed with sterile gauze. This must be done carefully after passing a small retractor, and is best done in the theatre. If the haemorrhage still persists there is no alternative to taking the patient to the theatre and, under an anaesthetic, passing a small vaginal retractor, washing out the vagina and inserting a mattress suture around the bleeding point. This is very rarely required, but emphasis must be put upon early recognition. If the nurse reports the first sign of pink discharge there will be few cases in which it is necessary to do more than give antiseptic douching.

(h) Results

Enterocoele

It is rarely that a patient with genital prolapse is not completely cured by means of a colporrhaphy no matter what her age or parity. In a series of cases investigated some considerable time after the operation, it was found that less than one per cent of those who had not subsequently had children had required any further operation. Recurrence of prolapse of the uterus is very rarely seen. Those who have trouble almost invariably have some bulging of the upper part of the posterior vaginal wall, in some cases a true enterocoele, in others a bulging of the pouch of Douglas attached to the vaginal wall. In these cases the deep suturing of the pelvic floor has been deficient and the hernia at the base of the pouch of Douglas has not been closed. Care in this part of the operation will prevent this type of recurrence, but it is the most difficult part of the operation and in a few cases, even when great care is taken, this gap is not closed.

Treatment of recurrence.—Treatment of this recurrence requires removal of the redundant skin, dissection of the peritoneum lining the sac, and closure of the gap in the floor of the pelvis by deep suturing of the muscles and parametrium at the base of the utero-sacral ligament. To obtain good access it may be necessary to split the perineum, when the operation becomes again a colpo-perineorrhaphy.

Stress incontinence

Sling operation for stress incontinence

In some cases of stress incontinence there has been so much damage to the anterior pelvic floor, through which the urethra passes, that it is difficult to stitch sufficient of this structure over the urethra in order to give perfect sphincter action. Because of this Millin and Read (1948) and others have devised operations whereby a strip of abdominal fascia on each side is dissected from the subjacent muscle except for the distal end. The abdomen is opened in the midline below the reflexion of the peritoneum: the pubovesical space is opened: the urethra is freed and the strips of fascia are passed under it and united. In this way a sling of fascia, with the ends attached to the abdominal muscles, is placed around the urethra. When the muscles contract, as they do in coughing or straining (the conditions which produce stress incontinence), the fascial sling around the urethra is tightened and so acts as a sphincter.

There may be a few cases requiring such an operation, but in the hands of a competent and careful vaginal operator they will be few.

Failure of an anterior colporrhaphy to cure stress incontinence usually means that the bulging urethra has not been sufficiently separated from the adjacent musculature to allow enough of this to be plicated over the urethra to renew the sphincter action. These sling operations are not described here in

detail as they are rarely required, and if they are they should be carried out by an expert.

Cases of dystocia following amputation of the cervix have been described, *Dystocia* and for this reason some operators are shy of removing the cervix. Carried out properly this operation rarely, if ever, causes dystocia; much more frequently subsequent labours are short, because there is not so much cervix to dilate.

For the same reason there is a slightly increased tendency to miscarry or to *Miscarriage* have premature labour; it calls for a little extra care during the pregnancy if the patient shows any sign of such an occurrence.

REFERENCES

Millin, T., and Read, C. D. (1948) *Post Grad. med. J.*, 24, 3, 51.

Shaw, W. F. (1933) *Amer J Obstet. Gynec*, 26, 667

— (1934). *J Obstet Gynaec*, 41, 853

[References to other titles are given under Pelvic Organs—Displacement, in the Index Volume]

PEPTIC ULCER AND ITS COMPLICATIONS

BY SIR HENEAGE OGILVIE, K.B.E., M.D., M.Ch.,
F.R.C.S., HON.F.A.C.S., HON.F.R.C.S.(C.), HON.F.R.A.C.S.,
HON.M.S. FOUAD I

SURGEON, GUY'S HOSPITAL; SURGEON, ROYAL MASONIC HOSPITAL; MAJOR-
GENERAL, ARMY MEDICAL SERVICE

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1. DEFINITION

262.] Peptic ulcers occur only in parts of the alimentary canal exposed to peptic digestion. They are usually limited to the stomach and first part of the duodenum, but they may be found in situations to which pepsin and hydrochloric acid are abnormally introduced, by sphincteric deficiency (as in the lower end of the oesophagus), by aberrant secretion (as in Meckel's diverticulum containing ectopic gastric mucosa), or by operation (as in the jejunum after a short-circuiting operation). *Exposure to peptic digestion*

2. AETIOLOGY

Peptic ulcers never occur naturally in animals. Acute ulcers can be produced by every kind of injury to the upper alimentary tract, but chronic ones can be produced only by insults so gross that they bear no relationship to clinical

*Value of
animal
experimenta-
tion*

experience. Animal experimentation in this field, dealing as it does with creatures differing profoundly from men in their habits, diet and digestive apparatus, is in any case a fallacious guide to human pathology. Clinical observation and reports from many countries make it clear that peptic ulcers occur in man in quite unrelated conditions, and that their causes are neither single nor simple.

*Factors
relating to
peptic ulcer*

The following factors have been proved to bear a relationship to the formation of peptic ulcers.

(1) Diathesis

The worrying, anxious type of patient is prone to peptic ulceration, particularly of the duodenum, and the diathesis and the ulcers tend to run in families. Davies and Wilson (1937, 1939) have investigated the relationship of psychological upsets to the origin and recurrence of ulcers. Wolf and Wolff (1943) have shown by direct observation that anxiety and hostility increase the blood flow of the gastric mucosa.

(2) Acid

Ulcers occur naturally only when there is free hydrochloric acid. They may be produced in animals by increasing the acid in its normal site or by diverting it to a fresh site, and they may be made to heal by removing the acid.

(3) Chemical substances

Some substances, such as aspirin and alcohol, by acting on the mucous membrane locally, some, such as histamine and probably tobacco, by increasing the output of acid, and some, such as cinchophen, gastrototoxic serum and the toxins of uraemia, by damaging the mucosa through the blood stream, may be responsible for, or contribute to, the formation of ulcers.

(4) Nervous impulses

Stimulation of the midbrain may cause ulcers in animals, apparently by promoting increased secretion. Cushing (1932) reported three fatalities from perforated peptic ulcer after operations on the base of the brain.

(5) Infection

Rosenow (1923) and many others have produced ulcers in animals by the injection of organisms. In man, acute duodenal ulcers may follow appendicitis, burns and septic wounds.

(6) Local trauma

Ulcers occur where the stomach wall is compressed by the margin of a diaphragmatic hernia or by a band. Acute duodenal ulcers sometimes occur as a result of a severe blow on the abdomen. Duodenal, jejunal and experimental ulcers tend to occur $\frac{1}{2}$ inch beyond the opening from which the gastric efflux is ejected.

(7) Malnutrition

Chronic malnutrition, of which protein deficiency is probably the most important cause, has been shown to be a cause of peptic ulceration in

prisoners in camps in Germany and Japan, and among the natives of the ulcer belt in Southern India.

In European countries it is becoming increasingly clear that duodenal and gastric ulcers have a different aetiology. Duodenal ulcers are seen preponderantly in men of the intellectual, worrying, anxious type, and attacks are precipitated by overwork and heavy smoking; gastric ulcers occur with equal frequency in both sexes, in patients of the listless, unambitious type with an acid secretion well below the average level, and malnutrition and oral sepsis appear to be related factors in such cases. In Southern India, ulcers which are usually more extensive and scarred than those of similar duration seen in Europe, but which in other respects show no essential differences, are probably due to protein deficiency and the use of strong peppers to render the monotonous fare more appetizing.

Varying aetiology

Related factors

It seems clear that hydrochloric acid, although it governs the site where ulcers occur and determines their persistence, is not the cause of their origin. All animals and all healthy men have a high acid level; animals, however, do not develop ulcers, and of the men who do many have a low acid curve. What distinguishes the ulcer patient is not the height, but the duration, of acid secretion. Acid is secreted continuously, not merely before and during meals, but between them and during sleep. The cause of this continuous secretion is possibly hormonal rather than vagal; such a view brings the ulcer diathesis into line with other psychosomatic diseases characterized by hormonal imbalance, glycosuria, hypothyroidism and hypertension. Acute ulcers may occur in any part of the mucous lining of the body just as they occur on any part of the cutaneous surface or the visible mucous membranes, but whereas in other places they heal, in the presence of hydrochloric acid they may be digested away and become peptic ulcers.

Duration of acid secretion

3. PATHOLOGY AND MORBID ANATOMY

Evidence of healed peptic ulceration is found at from 10 to 12 per cent of all necropsies. The proportion of duodenal to gastric ulcers is about four to one.

(1) Gastric ulcer

A chronic gastric ulcer probably starts as an acute ulcer. Acute ulcers are usually multiple. When seen in the stomach at necropsy, by gastroscopy or occasionally at operation when the stomach has been opened to search for the cause of an unexplained haematemesis, they may be found at any part. They appear first as haemorrhagic patches and later as sloughs involving the whole or part of the mucous membrane. They usually go no deeper than the muscle coat; if they do they may perforate. The majority heal without leaving a scar.

Initial appearance

Chronic ulcers are rarely seen except on the lesser curvature. The reason why acute ulcers in the fundus or the anterior and posterior walls usually heal is probably because in these situations the mucous membrane is folded and loosely attached to the outer coats, whereas on the lesser curve it is smooth, relatively immobile and fixed. Chronic ulcers are usually single. They may be found on any part of the lesser curve, but adjacent to the cardia and in the pre-pyloric inch they are rare. Two-thirds of them are found between 2 and 4 inches from the pylorus. When first discovered they are usually from $\frac{1}{2}$ inch

Chronic ulcers rare

Site

to 1 inch in diameter. Up to this size they are circular, but with further extension they become saddle-shaped, spreading on to the posterior and, to a lesser extent, on to the anterior wall adjacent to the lesser curve. Seen in the opened stomach or through the gastroscope they appear as circular craters, with a yellow sloughing base and thick rounded purple edges towards which folds on the anterior and posterior walls converge (Plate III).

Inflammatory reaction

A gastric ulcer is first a mucosal lesion, but it deepens by peptic digestion and penetrates the muscularis mucosae and muscular coats. As the base of the ulcer deepens, there is an inflammatory reaction beneath it, so that the deepest part consists of oedematous granulation tissue and acute perforation is rare. The arteries of the lesser curvature become involved in the base of the ulcer, but they usually thrombose before the ulcer crater reaches them, so that severe haemorrhage is uncommon. With further extension, adjacent organs are invaded; in almost 20 per cent of cases the floor of the ulcer is formed by the pancreas.

Hour-glass deformity

A gastric ulcer heals by the normal processes of repair. Granulation tissue becomes fibrous tissue, fibrous tissue contracts, approximating the edges of the defect in the muscular coats, and a single layer of epithelium grows across the small gap that remains. The larger the ulcer and the longer it has remained unhealed, the slower will be the process of repair and the more readily will it break down again. A large saddle-shaped ulcer of the lesser curve may, by its cicatrization, give rise to an hour-glass deformity of the stomach; this complication is virtually limited to women.

It is said that old-standing gastric ulcers may become cancerous, but that this occurs at all frequently is open to question. The evidence that they do so is entirely *post hoc* and based on histology. In a proportion of stomachs removed for what is thought to be gastric ulcer (one that varies from 5 per cent in British experience to some 40–50 per cent in that of some American hospitals) the pathologist reports that the ulcer shows cancerous transformation at some point; the same evidence might be interpreted as showing that a very scirrhus gastric cancer had been extended at one edge by peptic digestion. Clinical evidence points the other way. Of patients with typical gastric ulcers with remission, treated medically and carefully followed up, hardly one in two hundred dies of cancer, and of patients with ulcer-cancers, diagnosed as such after removal, very few will be found to have a history of over 4 years or of starting before the age of 40.

(2) Duodenal ulcer

Chronic duodenal ulcers also probably start as acute lesions, but acute duodenal ulcers are rarely recognized until they perforate. Nevertheless, those cases diagnosed on clinical and radiological grounds as "duodenitis" or "duodenal spasm" may be examples of acute mucosal erosions.

Constant position

Perforating duodenal ulcers and early chronic ulcers are found in a constant position—the anterior wall from $\frac{1}{4}$ to $\frac{3}{4}$ of an inch beyond the pylorus. Gastric ulcers are usually single, whereas duodenal ulceration, when it persists, is apt to give rise to multiple lesions, the commonest example being a second posterior ulcer, originating apparently by contact with the anterior one. Later there may be several confluent ulcers and the whole of the first part of the duodenum becomes grossly scarred and distorted.

The process of extension of a duodenal ulcer is essentially similar to that of a gastric ulcer, but it differs in two particulars. First, the reaction that precedes the advancing ulcer and the adhesions that bind it to neighbouring structures are not so complete, so that perforation may occur even in old-standing cases. Secondly, when a posterior ulcer, eroding the pancreas, reaches the gastro-duodenal artery it is apt to give rise to catastrophic haemorrhage. The repair of duodenal ulceration may lead to pyloric stenosis, but cancer never supervenes.

Differences in processes of ulcer extension

(3) Coexistent gastric and duodenal ulceration

Hurst (1929) encountered a combination of ulcers of both types in 21 cases in a series of about 400. The usual sequence is that of an old-standing duodenal ulcer going on to stenosis, and a gastric ulcer arising as a result of the retention and subsequent gastritis.

4. CLINICAL PICTURE

Acute gastric and duodenal ulcers are not unknown in infancy, but, apart from this, peptic ulcers of either variety are very rare in the first decade of life and uncommon until the close of the second decade. The majority appear between the ages of 18 and 35. The appearance, for the first time, of a genuine peptic ulcer after the age of 40 is unusual, and investigation in such cases will often show either that there have been previous bouts of indigestion suggesting ulceration, or that the ulcer is malignant and not peptic.

Age incidence

Many patients give a history that other members of the family have suffered similarly. The worrying, anxious outlook of the typical victim of duodenal ulcer is often inherited and later is aggravated by parental example.

Familial predisposition

Gastric and duodenal ulcers, in most respects, are different diseases, but they have certain features in common: these are, that they first occur in adolescence and early adult life, that their periods of activity recur in bouts at first lasting for from 2 to 5 weeks and separated by several months of freedom, but with each repetition tending to last longer, to take a severer form, and to recur sooner; and that the leading symptom is pain, of a dull boring character, which bears a definite time-relation to a preceding meal, and which assumes a similar site and time-table at each recurrence.

(1) Gastric ulcer

Gastric ulcers are seen with equal frequency in both sexes. Some patients are of the anxious duodenal type, but the majority are placid, even listless.

Sex incidence

Pain, which is the symptom that brings the patient to the doctor, takes the form of a deep-seated ache, usually felt in the midline about half-way between the xiphisternum and the transpyloric plane, but very vaguely located and described. Radiation is unusual, except in the case of deep penetrating posterior ulcers when the pain may be referred to the back, the left scapula or the left shoulder. The time of onset is said to vary with the site of the ulcer, high ulcers giving rise to pain about half an hour after food, ulcers near the pylorus causing pain some 2 hours later; the time-relations of the pain due to gastric ulcer are not, however, usually as regular as those of the pain due to

The nature of the pain

duodenal ulcer. Gastric ulcer pain dies away spontaneously and it does not wake the patient at night.

Vomiting

Vomiting is a frequent accompaniment of gastric ulcer, and usually relieves the pain immediately. Moderate haematemesis occurs in about 20 per cent of cases, but really severe ones are rare; *melaena* without haematemesis is very uncommon.

A subtype of gastric ulcer occasionally seen is a clinically silent and rapidly developing ulcer occurring in men and women aged 60 years or more. The site is high on the lesser curve, and the ulcer is often an inch or more in diameter when it is first discovered. There is usually little pain, and the ulcer is shown by radiography undertaken in investigation of an unexplained indigestion. Cancer is, and should be, suspected owing to the age of the patient and the size of the lesion, but the crater heals rapidly with the usual methods of treatment, in which rest in bed is undoubtedly the most important item.

Radiographic diagnosis

(2) Duodenal ulcer

Sex incidence

Duodenal ulcers preponderate in men in the proportion of about four to one. The patients are usually intelligent, ambitious and hard-working individuals who worry more than usual or have more than usual to worry about; in addition they are, in most cases, heavy cigarette smokers.

The nature of the pain

The pain is usually more severe than that of gastric ulcer; it is also more constant in its onset—that is, it occurs after every meal and at the same time after every meal. It repeats the same time-table in subsequent attacks. The nature of the pain is no better described than that of gastric ulcer; it is deep, boring and severe, being likened to toothache or cramp, seldom to hunger pain, a favourite simile of the surgical text-book. Its site is more accurately localized than that of gastric ulcer, usually on the transpyloric plane slightly to the right of the midline. Pain is seldom referred and hyperaesthesia is unusual.

Pain sequence in the two types of ulcer

Duodenal pain never appears till some time after a meal, usually 2 hours later, but sometimes so much later that the patient describes it as preceding the following meal rather than as following the preceding one. The pain persists till the next meal is taken; thus, while the rhythm of gastric ulcer is food, pain, comfort, food, that of duodenal ulcer is food, comfort, pain, food. Duodenal pain often comes on at night, waking the patient between 2 a.m. and 4 a.m.

Vomiting

Vomiting seldom occurs spontaneously in duodenal ulcer but when it does it relieves the pain; self-induced vomiting, therefore, is frequently described. Moderate haematemesis is more rare, but severe and dangerous haematemesis is much more common than is the case with gastric ulcer. *Melaena*,

by haematemesis, followed later by *melaena*, and by ...

Two clinical subtypes of duodenal ulcer are seen: the acute perforating ulcer, and the silent bleeding ulcer of the middle-aged man.

About 50 per cent of duodenal perforations occur in men who have had no previous ulcer pain; men, indeed, who have been well up to the time of the catastrophe or at most have felt unwell for only a few days. These ulcers, as seen at operation, have all the characteristics of an acute lesion, and not more than half of them show any tendency to recur.

Severe haematemesis and melaena, indicating a loss of from 2 to 4 pints of blood in the space of a few hours, is seen not infrequently in men between the ages of 40 and 55 who have not been known to have had a duodenal ulcer before. Their history has not been silent, but they have suffered from bouts characterized by vague digestive disturbances, by a feeling of fullness after meals and by a frequent desire to belch. Pain has been inconspicuous, and felt in the back rather than the abdomen. The ulcer is usually a posterior one eroding the pancreas.

(3) Progress

With the passage of time, as the recurring lesion becomes more fibrous and more fixed, the clinical picture of both gastric and duodenal ulcers becomes modified in two directions; the attacks become more frequent and severe, and symptoms referable to stenosis make their appearance. *Modification of clinical picture*

Gastric ulcers, no less than duodenal ulcers, can be healed in their early stages by medical treatment, but when their base has become fibrous, particularly when it becomes fixed to surrounding structures, permanent cure is unlikely and relapses occur as soon as the patient attempts to return to a normal diet. The final state is one of chronic invalidism and constant pain.

Malignant changes may occur in a true gastric ulcer, but they are so rare that their possibility should not be allowed to influence treatment. Cicatrization, in an organ so large as the stomach, is rarely enough to produce stenosis; the healing of a large saddle-shaped ulcer of the lesser curve, however, may produce an hour-glass stomach, the symptoms of which are indistinguishable from those of pyloric stenosis following a duodenal ulcer. *Rare malignant changes*

A duodenal ulcer, once treated, can remain healed if the factors that first caused it can be eliminated—a rare event with patients of the duodenal type. Recurrences are usually brought about by fresh worrying and further orgies of smoking. Once permanent deformity of the duodenum has been established, freedom from symptoms can be secured only by constant dieting and a state as miserable as that of the gastric ulcer patient is reached, though usually after a longer period of intermittent freedom.

The onset of pyloric stenosis is usually heralded by a lessening of the pain as the muscles, the contractions of which have caused it, tire out. Infrequent vomiting of large amounts of material, often having a foul smell and often containing food residue several days old, is the typical symptom, accompanied by those of inadequate nourishment—thirst, loss of weight and constipation. *The onset of pyloric stenosis*

(4) Physical examination

The physical signs that can be found in patients with peptic ulcer are seldom as helpful as is the history. Tenderness may be found over the duodenum, but is less often found over the site of a gastric ulcer. Abdominal guarding and hyperaesthesia are quite exceptional. When pyloric or mid-gastric stenosis is present, the dilated stomach can usually be outlined by percussion, and a succussion splash can be elicited by shaking the patient. It must be remembered that a similar succussion splash may be audible when a normal stomach is shaken, up to 2 hours after a liquid meal. *Succussion splash*

GASTROSCOPY



(a)



(b)



(c)



(d)

The progress of healing of an ulcer of the lesser curve of the stomach, as seen through the gastroscope. The drawings were made at intervals of a fortnight. In (a) the ulcer is viewed obliquely. In (b) the crater is smaller and radiating folds are appearing as a result of the diminished circumference. In (c) the gastroscope is close to the edge of the crater and so there is some magnification of the image, in reality the crater is much smaller than it appears. In (d) folds are seen to radiate from a central scar. (By courtesy of Mr. Norman Tanner.)

PLATE III

chronic to demand surgery may be inferred even though an ulcer cannot be demonstrated.

(2) Fractional gastric analysis

The fractional test-meal may help in the diagnosis of peptic ulcer, and also in the decision on the type of operation to be performed in a particular case. *Fractional test-meal*

In duodenal ulcer the resting juice is abundant (60 or more cubic centimetres), showing a high level of total and free hydrochloric acid. After an initial fall when the meal is swallowed, the curve is typically a rising one—the level of both total and free acid running parallel at a height well above the highest normal (55 and 45 cubic centimetres N/10 NaOH in Ryle's charts)—and ends at its highest point, instead of on a downward slope as in a normal test-meal. The stomach may retain the meal for the normal time (2½ hours), or may empty more rapidly (Fig. 288).

In gastric ulcer the test-meal is often within normal limits; variations are usually in the directions of hypochlorhydria and delayed emptying. In both gastric and duodenal ulcers blood will be found in the test-meal if the ulcer is active at the time of examination. *Hypochlorhydria and delayed emptying*
Evidence of malignancy

(3) Examination of the faeces for occult blood

The discovery of occult blood in the stools, after administration of a meat-free and chlorophyll-free diet for 4 days, during which the bowels have been opened daily, is proof of bleeding somewhere in the alimentary tract. Occult blood is present when a peptic ulcer is active, and absent when it is quiescent or healing. The persistence of blood after a patient with a supposed peptic ulcer has been on strict medical treatment for a month offers strong presumptive evidence that the lesion is in fact malignant.

(4) Gastroscopy

Direct inspection of the interior of the stomach through a flexible gastroscope, by one experienced in the method, gives information of the greatest value, second only to that afforded by radiography. Gastroscopy is chiefly needed by the surgeon:

(a) to determine whether a gastric lesion, that from its history, symptoms or radiological appearances is not quite typical of either condition, is a peptic or a malignant ulcer;

(b) to study the progress of healing in a gastric ulcer under prolonged treatment (Plate III);

(c) to seek for an ulcer suspected on clinical grounds, usually because of repeated haematemesis, but not demonstrable radiologically; and

(d) to decide, in the case of an intractable duodenal ulcer, whether a short-circuiting operation will suffice or whether the gastric mucosa is of the rugose high secretive type, in which case radical surgery is advisable.

6. DIFFERENTIAL DIAGNOSIS

(1) Distinction between gastric and duodenal ulcer

The characteristics of the patient affected, the history and the x-ray appearances will usually allow of a correct diagnosis. Gastric ulcers penetrating the lower third of the lesser curve and infiltrating the lesser omentum, however,

may mimic the symptoms and x-ray appearances of a duodenal ulcer. The distinction is in any case unimportant to the surgeon, whose need is to determine that the lesion is a peptic ulcer and whether it is refractory to medical treatment.

(2) Distinction between peptic ulcer and other causes of pain or indigestion

(a) Cancer of the stomach

Most gastric cancers are silent, but those near the pylorus may give a history resembling that of ulcer. The age of the patient, the variable quality of the pain and the failure of food, alkalis or vomiting to relieve it, the progressive nature of the symptoms and the persistence of occult blood in spite of treatment, the loss of appetite and the wasting usually raise suspicions of malignancy. A skiagram, followed in cases of doubt by gastroscopy, will usually settle the diagnosis.

(b) Pylorospasm without a lesion

Disturbance
of neuro-
muscular
mechanism of
gastric
digestion

Worry and smoking may give rise to recurring pain with all the characters of duodenal pain, and similarly relieved by food. Gall-stones, subacute or chronic appendicitis, and many other lesions in the abdominal cavity may, by a visceral reflex, upset the neuro-muscular mechanism of gastric digestion and also give a clinical picture resembling that of duodenal ulcer.

(c) Other pains related to meals

The taking of a meal is the signal for renewed peristalsis throughout the alimentary canal. Inflammatory lesions in this territory, such as regional ileitis and subacute appendicitis, or in its neighbourhood, such as tubal infection, thus may be signalized by pain the maximal intensity of which is related to meals.

(3) The differential diagnosis of haematemesis

Not more than about 60 per cent of haematemeses are due to peptic ulcer. Many of the more severe haemorrhages are unexplained, since a lesion is not discovered even at necropsy; such haemorrhages are recorded as being due to "gastrostaxis". Many are due to multiple acute mucosal erosions of the stomach which are possibly infective in origin. Others may be due to the action of toxins, such as those of uraemia, or of drugs among which aspirin must be included, or to the rupture of oesophageal varices in portal cirrhosis or of varicose vasa brevia in splenic enlargement, to blood diseases such as thrombocytopenic purpura and haemophilia, to polypoid tumours, or—uncommonly—to cancer.

"Gastrostaxis"

(4) The differential diagnosis of melaena

When melaena follows a history of duodenal ulcer, its origin is unmistakable. When it is the only symptom duodenal ulcer still remains the most probable cause, but a peptic ulcer in a Meckel's diverticulum or an early cancer of the colon may be the source of the blood.

7. PROGNOSIS

Medical
treatment

Both gastric and duodenal ulcers can be cured by medical treatment, provided that the treatment is instituted early and continued till the ulcer is well healed.

and provided too that the factors which caused it can be removed afterwards. Both varieties tend to relapse when these requirements are not fulfilled, to become refractory, and eventually to lead to a state of chronic invalidism or to death from complications. The complications of gastric ulcer are perforation, haematemesis, stenosis and malignant change. Perforation, major haemorrhage and stenosis are all more common in duodenal ulcer than in gastric ulcer, but cancer never occurs in duodenal ulcer. *Complications*

It is estimated that about one person in ten of the adult population of Great Britain suffers at some time from peptic ulceration, and about four thousand are reported as dying annually from this cause; since, however, each of the above possibly fatal complications may be recorded under some other heading in the death certificate, it is impossible to arrive at a useful estimate of the over-all mortality. The analysis of individual figures is of no great value, for they are always good—otherwise they would not be published; personal statistics are the basis of much unsound teaching. The aggregated records of a hospital are more reliable, but these too are influenced by locality, by transport facilities, by adequacy of beds, and by the interest of the staff (and that of neighbouring institutions) in gastric problems. The following figures from a non-teaching general hospital in a large town have therefore a very limited value. *Estimate of mortality*

(1) *Analysis of one thousand cases of peptic ulcer admitted*

Uncomplicated ulcer	-	-	-	Duodenal	450	
				Gastric	210	
						660
Perforation of peptic ulcer	-	-	-			60
Haemorrhage from peptic ulcer	-	-	-			215
Jejunal ulcer	-	-	-			65
						<u>1,000</u>

Thus in this hospital 27.5 per cent of the admissions were for the complications of peptic ulceration, and 6.5 per cent for complications of ulcer surgery.

(2) *Analysis of two hundred and ten cases of gastric ulcer*

(a) *Treated medically*

Hospital deaths	-	-	-	-	-	1 per cent
Condition of survivors followed up for 5-10 years						
Satisfactory	-	-	-	-	50	"
Unsatisfactory	-	-	-	-	40	"
Dead	-	-	-	-	10	"
					<u>100</u>	"

(b) *Treated surgically*

Hospital deaths	-	-	-	-	-	9 per cent
Condition of survivors followed up for 5-10 years						
Satisfactory	-	-	-	-	77	"
Unsatisfactory	-	-	-	-	13	"
Dead	-	-	-	-	10	"
					<u>100</u>	"

(3) *Analysis of four hundred and fifty cases of duodenal ulcer*(a) *Treated medically*

Hospital deaths	-	-	-	-	-	1 per cent
Condition of survivors followed up for 5-10 years:						
Satisfactory	-	-	-	-	-	30 "
Unsatisfactory	-	-	-	-	-	60 "
Dead	-	-	-	-	-	10 "
						100 "

(b) *Treated surgically*

Hospital deaths	-	-	-	-	-	5 per cent
Condition of survivors followed up for 5-10 years.						
Satisfactory	-	-	-	-	-	65 "
Unsatisfactory	-	-	-	-	-	25 "
Dead	-	-	-	-	-	10 "
						100 "

This last group may be resolved into:

Gastrectomy (36)

Hospital deaths	-	-	-	-	-	8 per cent
Of survivors.						
Satisfactory	-	-	-	-	-	82 "
Unsatisfactory	-	-	-	-	-	9 "
Dead	-	-	-	-	-	9 "
						100 "

Gastro-jejunostomy (114)

Hospital deaths	-	-	-	-	-	3.5 per cent
Of survivors.						
Satisfactory	-	-	-	-	-	60 "
Unsatisfactory	-	-	-	-	-	30 "
Dead	-	-	-	-	-	10 "
						100 "

Inference

are very much better (Fig. 289). Gastro-jejunostomy has a lower death-rate than gastrectomy, but also a lower rate of cure. The figures are not those of a surgical clinic particularly concerned with gastric surgery, as is shown by the three-to-one preponderance of gastro-jejunostomy over gastrectomy in the treatment of duodenal ulcer. There are many surgeons whose cases show a mortality over several years and over several hundred cases not exceeding 0.5 per cent for gastro-jejunostomy and 2 per cent for gastrectomy, and who can claim a percentage of satisfactory late results of well over 90.

Prognosis regarding haemorrhage

The prognosis of haemorrhage from peptic ulcer cannot be estimated with any greater accuracy than the total risk, because writers seldom define the amount of blood loss that qualifies a case for inclusion in their series. If haemorrhage is defined as the loss of an amount of blood sufficient to cause circulatory collapse—that is, 2 pints or more—the hospital mortality is about 10 per cent. If consideration is confined to a particular group, recurrent haemorrhage from a known chronic ulcer in patients of 50 or more years of

age with sclerotic arteries, the mortality is about 50 per cent. In such cases operative treatment carries a risk as well, but with an expert surgeon operating early in the haemorrhage, assisted by a good anaesthetist and transfusion service, it is not more than 5 per cent.

The prognosis with regard to perforation is even more difficult to assess. Perforations are duodenal in from 80 to 90 per cent of cases, but the proportion of duodenal ulcers that perforate varies very widely in different localities. Thus in Vizagapatam patients with perforations form only 2.2 per cent of

hospital admissions for peptic ulcer, whereas in Calcutta they form 22.4 per cent. The mortality varies greatly with the time between perforation and operation, and, therefore, with the district in which the hospital presenting the figures is situated and the transport facilities that serve it. For patients reaching hospital within 8 hours of perforation the mortality is about 5 per cent, and for those arriving later about 10 per cent.

8. INDICATIONS FOR SURGICAL INTERVENTION

The indications for operation in duodenal and gastric ulcer differ to some extent. The chief causes of duodenal ulcer lie in the temperament of the patient—that is, in factors that surgery cannot control—and operation is required in the main to deal with complications. In gastric ulcer the causes of persistence, if not of origin, are for the most part in the stomach itself, and if medical treatment has not caused the ulcer to heal before it has reached a certain size and become adherent to surrounding structures, it is unlikely ever to do so more than temporarily.

The operative treatment of duodenal ulcer calls for a variety of procedures, the simple operations often being unsatisfactory and the radical ones often difficult, whereas gastrectomy for gastric ulcer is a standardized and straightforward procedure. The failures of gastric surgery all follow operations for duodenal ulcer, whereas a permanent cure can be expected with some confidence after an operation for gastric ulcer. Thus patients with duodenal ulcer should, if possible, be kept on medical treatment till they are 45 years of age or till they suffer from complications, whereas those with gastric ulcer should be advised to submit to operation if they have failed to respond to a year of

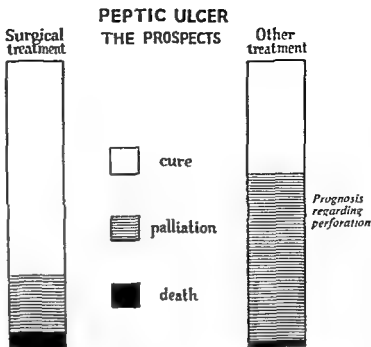


FIG. 289.—Diagrammatic representation of the place of surgery in the treatment of peptic ulceration.

(3) *Analysis of four hundred and fifty cases of duodenal ulcer*(a) *Treated medically*

Hospital deaths	-	-	-	-	-	1 per cent
Condition of survivors followed up for 5-10 years:						
Satisfactory	-	-	-	-	-	30 "
Unsatisfactory	-	-	-	-	-	60 "
Dead	-	-	-	-	-	10 "
						100 "

(b) *Treated surgically*

Hospital deaths	-	-	-	-	-	5 per cent
Condition of survivors followed up for 5-10 years:						
Satisfactory	-	-	-	-	-	65 "
Unsatisfactory	-	-	-	-	-	25 "
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Of survivors.						
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Unsatisfactory	-	-	-	-	-	30 "
Dead	-	-	-	-	-	10 "
						100 "

Inference

From these particular figures it is seen that the hospital mortality of surgical treatment exceeds considerably that of medical treatment, but the late results are very much better (Fig. 289). Gastro-jejunostomy has a lower hospital death-rate than gastrectomy, but also a lower rate of cure. The figures are not those of a surgical clinic particularly concerned with gastric surgery, as is shown by the three-to-one preponderance of gastro-jejunostomy over gastrectomy in the treatment of duodenal ulcer. There are many surgeons whose cases show a mortality over several years and over several hundred cases not exceeding 0.5 per cent for gastro-jejunostomy and 2 per cent for gastrectomy, and who can claim a percentage of satisfactory late results of well over 90.

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The prognosis with regard to perforation is even more difficult to assess. Perforations are duodenal in from 80 to 90 per cent of cases, but the proportion of duodenal ulcers that perforate varies very widely in different localities. Thus in Vizagapatam patients with perforations form only 2.2 per cent of

hospital admissions for peptic ulcer, whereas in Calcutta they form 22.4 per cent. The mortality varies greatly with the time between perforation and operation, and, therefore, with the district in which the hospital presenting the figures is situated and the transport facilities that serve it. For patients reaching hospital within 8 hours of perforation the mortality is about 5 per cent, and for those arriving later about 10 per cent.

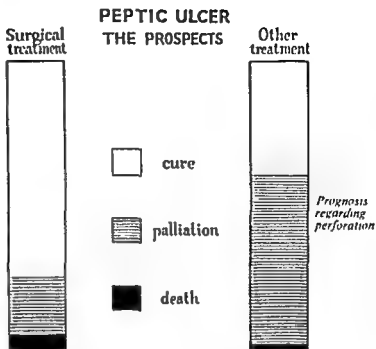


FIG. 289.—Diagrammatic representation of the place of surgery in the treatment of peptic ulceration

8. INDICATIONS FOR SURGICAL INTERVENTION

The indications for operation in duodenal and gastric ulcer differ to some extent. The chief causes of duodenal ulcer lie in the temperament of the patient—that is, in factors that surgery cannot control—and operation is required in the main to deal with complications. In gastric ulcer the causes of persistence, if not of origin, are for the most part in the stomach itself, and if medical treatment has not caused the ulcer to heal before it has reached a certain size and become adherent to surrounding structures, it is unlikely ever to do so more than temporarily.

The operative treatment of duodenal ulcer calls for a variety of procedures, the simple operations often being unsatisfactory and the radical ones often difficult, whereas gastrectomy for gastric ulcer is a standardized and straightforward procedure. The failures of gastric surgery all follow operations for duodenal ulcer, whereas a permanent cure can be expected with some confidence after an operation for gastric ulcer. Thus patients with duodenal ulcer should, if possible, be kept on medical treatment till they are 45 years of age or till they suffer from complications, whereas those with gastric ulcer should be advised to submit to operation if they have failed to respond to a year of

(3) *Analysis of four hundred and fifty cases of duodenal ulcer*(a) *Treated medically*

Hospital deaths	-	-	-	-	-	1 per cent
Condition of survivors followed up for 5-10 years:						
Satisfactory	-	-	-	-	30	"
Unsatisfactory	-	-	-	-	60	"
Dead	-	-	-	-	10	"
					100	"

(b) *Treated surgically*

Hospital deaths	-	-	-	-	-	5 per cent
Condition of survivors followed up for 5-10 years:						
Satisfactory	-	-	-	-	65	"
Unsatisfactory	-	-	-	-	25	"
Dead	-	-	-	-	10	"
					100	"

This last group may be resolved into:

Gastrectomy (36)

Hospital deaths	-	-	-	-	-	8 per cent
Of survivors						
Satisfactory	-	-	-	-	82	"
Unsatisfactory	-	-	-	-	9	"
Dead	-	-	-	-	9	"
					100	"

Gastro-jejunostomy (114)

Hospital deaths	-	-	-	-	-	3.5 per cent
Of survivors:						
Satisfactory	-	-	-	-	60	"
Unsatisfactory	-	-	-	-	30	"
Dead	-	-	-	-	10	"
					100	"

Inference

From these particular figures it is seen that the hospital mortality of surgical treatment exceeds considerably that of medical treatment, but the late results are very much better (Fig. 289). Gastro-jejunostomy has a lower hospital death-rate than gastrectomy, but also a lower rate of cure. The figures are not those of a surgical clinic particularly concerned with gastric surgery, as is shown by the three-to-one preponderance of gastro-jejunostomy over gastrectomy in the treatment of duodenal ulcer. There are many surgeons whose cases show a mortality over several years and over several hundred cases not exceeding 0.5 per cent for gastro-jejunostomy and 2 per cent for gastrectomy, and who can claim a percentage of satisfactory late results of well over 90.

Prognosis regarding haemorrhage

The prognosis of haemorrhage from peptic ulcer cannot be estimated with any greater accuracy than the total risk, because writers seldom define the amount of blood loss that qualifies a case for inclusion in their series. If haemorrhage is defined as the loss of an amount of blood sufficient to cause circulatory collapse—that is, 2 pints or more—the hospital mortality is about 10 per cent. If consideration is confined to a particular group, recurrent haemorrhage from a known chronic ulcer in patients of 50 or more years of

(iii) Because cancer is suspected. The likelihood of malignancy must be borne in mind and operation advised when a gastric ulcer lies in the pre-pyloric inch, when an ulcer appears with a short progressive history in a patient over 45 years of age, when the crater is more than an inch in diameter, or when occult blood persists in the stools after a month of strict medical treatment.

(c) Contra-indications to continuing other forms of treatment

Operation may be indicated when there is an inability on the part of the patient, after several courses of treatment, to continue further—owing to temperament, occupation, circumstances or the need to travel in the future.

9. PRE-OPERATIVE MANAGEMENT

Operations for peptic ulcer, except those undertaken for the emergencies of perforation and haemorrhage, should be preceded by a period of careful preparation, with the two-fold purpose of making the patient fit for the operation and making the operation easy for the surgeon.

When patients come to operation during a period of quiescence, they merely need the preparation required for any major abdominal operation, particularly training in the breathing and leg exercises which they will be required to undertake as soon as they recover consciousness.

Training in breathing and leg exercises

A patient whose ulcer has been active lately may be suffering from alkalosis, owing to over-enthusiastic medication with alkalis, from dehydration, from anaemia or from protein deficiency. These errors and deficiencies must be corrected before any operation is undertaken, and they should, if possible, be corrected gradually during a period of several weeks in bed under the care of the staff who will nurse the patient afterwards. Alkalosis and dehydration can be corrected by omitting alkalis and giving enough fluid to produce a daily output of at least 50 ounces of urine. Anaemia can be corrected by the administration of ferrous sulphate (3 grains 4 times a day) together with a high protein diet, but if the response is unsatisfactory or the haemoglobin level is below 50 per cent, transfusion may be required. A haemoglobin reading of at least 70 per cent should be reached before operation.

Correction of alkalosis and dehydration

The ulcer itself should be healed, or at any rate quiescent, before operation is undertaken. Oedema surrounding an active ulcer, whether it be a duodenal or a gastric ulcer, increases greatly the technical difficulty and therefore the danger of an operation; in the case of a duodenal ulcer it interferes with satisfactory invagination of the duodenal stump, and in the case of a gastric ulcer with control of the left gastric vessels and high division of the lesser curve of the stomach. Two or three weeks in bed, with complete rest and an hourly diet rich in proteins, will greatly reduce the size of an ulcer of the lesser curve, as seen in a skiagram or with the gastroscope. In the case of large ulcers and emaciated patients, a duodenal tube may be passed through the nostril, and the "Belsen drip"—consisting of dried milk 100 grammes, glucose 400 grammes and water to 1,500 millilitres—may be given through it, the total volume is administered over a period of 24 hours, in addition to further fluids and more palatable foods by mouth.

Danger of oedema

The "Belsen drip"

Gastric lavage should not be performed before operation unless there is stenosis, in which case the stomach should be washed out night and morning

Indication for gastric lavage

treatment conscientiously followed, or earlier if there is any reason to suspect that the ulcer may be malignant or undergoing malignant change.

The indications for surgical intervention may be absolute or relative.

(1) Absolute indications

(a) Perforation

A patient whose ulcer has perforated is likely to die unless the perforation is sutured. Most perforations are duodenal.

(b) Major haemorrhage

(i) Patients over 50 years of age with chronic ulcers that have given rise to one or more previous massive haemorrhages, and who have bled again and are still bleeding 24 hours after they have come under observation and treatment, run a 50 per cent risk of death from haemorrhage, and only a 5-10 per cent risk of succumbing to an emergency operation undertaken to arrest it.

(ii) Those who have had several large haemorrhages, but are not bleeding at the time, should be protected from further risks by an interval operation.

(iii) Those patients with constant minor haemorrhage from an ulcer, usually gastric, who lapse into an anaemic state in spite of repeated transfusion, should undergo operation before their condition deteriorates further.

(c) Stenosis

Once the contraction of a healing ulcer has caused such a degree of obstruction that the muscles behind it are tiring out, so that the barium meal shows a delay of 6 hours or over in emptying a large stomach in which the barium is diluted by retained gastric contents, and a fluid level, the patient is at the beginning of a period of slow starvation, and operation is essential. This degree of obstruction is a not uncommon sequel of recurring duodenal ulceration, when the scar narrows an already narrow part of the alimentary tract, but seldom follows gastric ulcer, in which it gives rise to hour-glass stomach, a deformity rarely seen except in women and seldom causing complete obstruction.

*Effects of
cicatrizization*

Severe cicatrization insufficient to cause much obstruction may present indications, though less important ones, for operation. Such cicatrized ulcers never heal permanently, and by their mechanical interference they make frequent reactivation of the ulcer inevitable.

(2) Relative indications

(a) Failure of medical treatment

Patients and physicians will differ as to what is failure, that is, the point at which it becomes clear that further treatment offers nothing better than a life of permanent invalidism.

(b) The prospect of inevitable failure

Failure may be judged to be inevitable for the following reasons:

(i) Because the ulcer has caused so much deformity that, even when it has healed, a return to a fuller diet and further activity will cause it to become active again.

(ii) In the case of gastric ulcer, because of penetration of, or firm adherence to, the pancreas or liver.

(iii) Because cancer is suspected. The likelihood of malignancy must be borne in mind and operation advised when a gastric ulcer lies in the pre-pyloric inch, when an ulcer appears with a short progressive history in a patient over 45 years of age, when the crater is more than an inch in diameter, or when occult blood persists in the stools after a month of strict medical treatment.

(c) *Contra-indications to continuing other forms of treatment*

Operation may be indicated when there is an inability on the part of the patient, after several courses of treatment, to continue further—owing to temperament, occupation, circumstances or the need to travel in the future.

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*Training in
breathing and
leg exercises*

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*Danger of
oedema*

*The "Belsen
drip"*

Gastric lavage should not be performed before operation unless there is stenosis, in which case the stomach should be washed out night and morning

*Indication
for gastric
lavage*

for 3 or 4 days to overcome the inflammation of the mucous membrane and to allow the muscle coats to recover some of their tone.

10. SURGICAL ANATOMY

The descriptions of the anatomy of the stomach and duodenum given in standard text-books should be familiar to the surgeon. In certain respects they are over-simplified.

(1) The arteries

The right gastric artery : The right gastric or pyloric artery is a mythical, or at least an exceptional, structure ; in its place is found a leash of small vessels, coming mostly from the

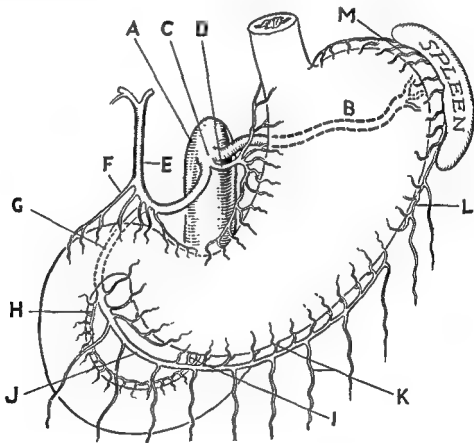


Fig. 290. The arteries of the stomach and duodenum. A: Aorta. B: Splenic artery, C: Common hepatic artery, D: Proper hepatic artery, E: Right gastric artery, F: Right gastroepiploic artery, G: Left gastric artery, H: Left gastroepiploic artery, I: Superior mesenteric artery, J: Inferior mesenteric artery, K: Splenic vein, L: Left gastroepiploic artery, M: Splenic vein.

and epiploic branches L: Left gastro-epiploic artery

hepatic artery, and spreading fan-wise to supply the upper border of the first inch of the duodenum and the distal fourth of the lesser curve of the stomach (Fig. 290).

The right gastric artery does not, as the figures show it, run across the left crus

below the oesophageal opening, and, after a short course as a trunk, breaks up some distance from the stomach into a number of branches, some of which go up towards the cardia, others down towards the pyloric antrum. The considerable interval between the main branches of the artery and the stomach wall is filled by a mass of fat and the terminal lymphatic glands of the lesser curve, the whole forming a gastro-pancreatic omentum that juts widely into the upper half of the lesser sac. Ligation and division of the main trunk of this artery, rather than of its branches, is an essential step in gastrectomy. The normal stomach is slung like a hammock between its two openings; the left gastric artery springs from the centre of this arc and is never pulled upon. The resected stomach hangs like a pendulum from the oesophagus and the peritoneal ligaments round the oesophageal opening; if the left gastric artery retains any attachments with the lesser curve it becomes an anchor, limiting movements.

The gastro-epiploic arch is once more a figment of anatomical imagination. The right and left gastro-epiploic arteries terminate close to each other in the greater curvature of the stomach, but they do not anastomose except through their omental branches. The gastric branches of the gastro-epiploic arteries leave the parent trunks about $\frac{1}{2}$ inch from the greater curvature of the stomach and are divisible into anterior and posterior branches each of which pierces the coats of the stomach very obliquely. The epiploic branches are the sole blood supply of the great omentum which receives nothing but trivial twigs from branches of the superior mesenteric artery.

Arterially as well as embryologically the first part of the duodenum belongs to the fore-gut. The upper and lower borders correspond to the lesser and greater curvatures of the stomach and receive, like them, a series of vessels in double rows. The right and left surfaces are—like the anterior and posterior surfaces of the stomach which they represent—free from vessels, and can be dissected from adhesions to surrounding structures or to the pancreas without fear of haemorrhage. After the bile papilla the duodenum becomes intestine, and receives vessels on its concavity only.

The anastomosis between adjacent vessels in stomach and duodenum is so free that there is little danger of sloughing, however many are tied. The theory of end-arteries, so beloved of the writer on the aetiology of ulcer, makes no sense to the surgeon. The boundary between the two viscera at the pylorus is, however, complete with regard both to lymphatics and blood-vessels; if after ligating all vessels down to the pylorus, any of the stomach is left behind, it will slough.

(2) The peritoneum

The upper leaf of the transverse mesocolon is always adherent, to a variable extent, to the posterior wall of the stomach, particularly at the pyloric end. Unless these adhesions are separated soon after the lesser sac is opened in the operation of gastrectomy, the middle colic artery may be divided in mistake for one of the gastric vessels.

11. THE AIMS OF ULCER SURGERY

Operations form one aspect only of the treatment of peptic ulcer. Some may be emergency operations undertaken to save life, but the majority are elective

Effective procedures

procedures designed to relieve a disease that is disabling but not necessarily fatal. The patient has pain and indigestion; he wishes to lose them, and he is prepared to face an operation if he is sure that it is reasonably safe and that its results are reasonably satisfactory. Ulcer surgery may have one, two or all of three objectives in view: the treatment of complications, the removal of the ulcer and the prevention of future ulceration.

12. THE INCISION

Midline incision

The two difficult parts of a gastric operation are dissection and closure of the duodenum, and ligation of the left gastric artery and division of the cardiac end of the lesser curvature. Right or left paramedian incisions which favour one hinder the other, and neither is high enough for easy access to the coeliac axis. Trans-rectus incisions are sound in theory and successful in practice when they are successful, but, when they fail, they are disastrous beyond remedy. The midline incision is the best. It is bloodless, it allows free access to the subdiaphragmatic space, it can be continued downwards through the umbilicus if need be, and upwards to the left of the xiphisternum, when it extends a good 2 inches higher than the paramedian incision can do; moreover, when it is closed by interrupted silk sutures, it gives a scar of great strength afterwards.

13. OPERATIONS FOR THE COMPLICATIONS OF ULCER

(1) Perforation

Gastric perforation rare

Gastric perforations are uncommon; the majority are duodenal, the hole is nearly always on the anterior wall $\frac{1}{2}$ inch beyond the pylorus, and in at least half the cases the ulcer that has perforated is an acute one.

Two-fold purpose of operation

Every perforation is a surgical emergency, but it does not necessarily demand an urgent operation. The purpose of operation is two-fold: to close the perforation, and to prevent what is at first a chemical peritoneal irritation from becoming a septic peritonitis.

Nature of exudate

A duodenal perforation is sealed off, temporarily at any rate, as soon as the gastric contents have escaped through it, being stuck by lymph to a neighbouring structure, usually the liver. At operation these adhesions must be separated before the hole can be seen and sutured. Suture is by no means a satisfactory watertight job, for the duodenal wall round the perforation is oedematous and friable, and the safety of the closure depends more on the post-operative treatment than the surgeon institutes than on the niceness of his needlework. The material that comes out through the perforation is acid and usually sterile, sometimes containing portions of food, but in most cases

the peritoneum can overcome the infection and absorb the material, provided that the contamination is not excessive or repeated, and provided that the natural mechanisms of repair are not hindered by injudicious meddling, of which needless drainage and unnecessary interference with movements of the diaphragm are the most common examples. Operation is under-

The material escapes first into the neighbourhood of the epiploic foramen and the right kidney pouch of Rutherford Morison, thence down the right paracolic gutter to the right iliac fossa, thence, by overflow, into the pelvis, and finally it floods the whole abdomen. Many patients are admitted in the first phase—that of limited soiling; the history is that of a perforation, but the general condition is excellent, the pulse rate is only moderately raised, and the tenderness and guarding are limited to the right hypochondrium and flank. The diagnosis can be confirmed by a skiagram, taken while the patient is in bed, which will show air under the diaphragm. Such patients do not always require operation—indeed, they may recover more quickly without it. As soon as the diagnosis is made the stomach is emptied by a tube passed through the nostril, and is, thereafter, kept empty by joining the tube to a suction apparatus. Treatment for peritonitis is instituted (*see Peritoneum and Peritonitis*, p. 550).

Patients admitted in the phase of limited soiling

When the material that has escaped into the peritoneum has flooded the greater part of the abdomen, recovery by natural means is possible, but a high

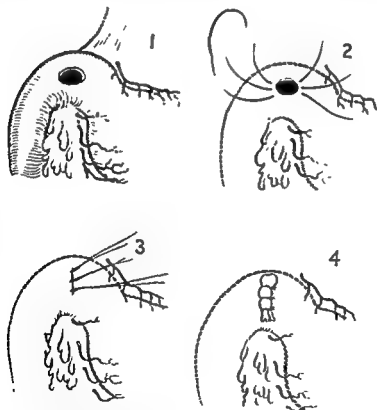


FIG. 291.—Closure of a perforated duodenal ulcer. (1) The perforation. (2) The sutures inserted (3) Sutures tied (4) Sutures tied again over a free omental graft.

morbidity and mortality from localized abscesses, particularly in the sub-phrenic spaces, is probable. In such cases operation should be advised. The abdomen is opened by a median incision, and the abundant free fluid which floods the field is removed by suction. If the amount is large, a $\frac{1}{2}$ -inch incision

Free fluid removed by suction

is made in the midline, above the pubis, on to a swab pushed down by the left hand in contact with the anterior abdominal wall; through this a second nozzle is introduced to empty the hypogastrium and pelvis while the operation is proceeding. As soon as excess fluid has been removed the stomach is wrapped in a moist gauze swab and drawn to the left by an assistant, while the operator separates the duodenum from the liver and brings the hole into view.

The hole is seldom more than a $\frac{1}{2}$ inch in diameter, but the duodenal wall round it is oedematous and friable for a further $\frac{1}{2}$ inch in all directions. Owing to this oedema, secure closure of the hole, and reinforcement by infolding the duodenal wall over it, is seldom possible, without an encroachment so wide that the lumen is narrowed—though the narrowing may be minimized by approximating the edges at right angles to the lumen (Fig. 291). It is better to be content with minimal approximation, and to rely on the removal of pressure from within and the natural sealing without to make the repair secure. Three stitches of fine chromicized catgut, on a small half-circle needle, are passed through all coats of the duodenal wall, one across the centre of the hole and one at each margin. These sutures are tied firmly, but not pulled so hard that they cut through, and are left long. A free graft of omental fat is laid over the closed hole, and the sutures are once again tied over it, and cut short.

A harmless antiseptic, such as 1 : 1,000 solution of 5-aminoacridine or sulph-anilamide powder, may be introduced into the peritoneum before the wound is closed. Drainage is seldom advisable.

A large hole in a previously scarred duodenum, the closure of which obviously perforation to symptoms, however, should be resisted. Emergency surgery should be as simple and rapid as possible, and to add fancy steps to the life-saving one of closure is bad judgement and bad surgery.

(2) Stenosis

Stenosis demanding treatment as such is usually at the pylorus, but mid-gastric stenosis, giving rise to hour-glass stomach, presents a similar problem. The treatment of stenosis *per se* is the establishment of a fresh passage, but since gastric and duodenal stenosis are sequelae of long-continued peptic

treatment of the stenosis.

If the patient is 50 or more years of age, if the history of ulcer is a long one, if the recent symptoms have been mechanical only and pain has been inconspicuous, and if the test meal shows a low acid curve after daily gastric lavage for 2 weeks, a short-circuiting operation may be undertaken with little hesitation. Many patients even of this group, however, recover their secretory power after the stasis has been overcome and eventually develop anastomotic ulcers, so that there is an increasing tendency to perform a more radical operation in all patients whose general condition does not raise their risk appreciably above the average.

Free graft of omental fat

Use of antiseptics

Possible need for gastro-jejunostomy or gastrectomy

Simple short circuit alone or with some radical procedure

Possible development of anastomotic ulcers

(a) *Short-circuiting operations for pyloric stenosis*

Many varieties of gastro-jejunostomy, and pyloroplasty have been devised; of these, only two can be recommended.

(i) *Posterior retrocolic short-loop gastro-jejunostomy* (Fig. 292).—After the abdomen has been opened and the diagnosis of uncomplicated pyloric stenosis has been confirmed, the transverse colon is drawn out of the wound and lifted up. A bloodless area is selected in the transverse mesocolon within the arch of the middle colic artery, and through this an opening is made into the lesser sac; this opening is enlarged with scissors to a slit about $2\frac{1}{2}$ inches

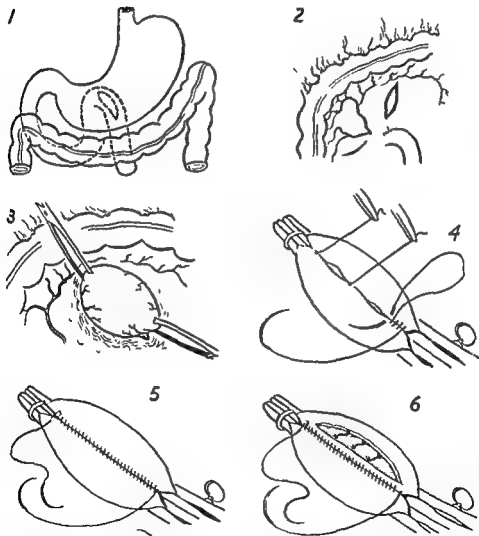


FIG. 292 (a) —Posterior retrocolic short-loop gastro-jejunostomy (1) Plan of the operation (2) Opening made in transverse mesocolon within arch of middle colic artery (3) Pouch of whole width of the posterior wall of the stomach is pulled through this opening (4) The pouch of stomach and the loop of jejunum are held together in a pair of twin clamps; two stay-sutures have been inserted, and the two-needle sero-muscular suture has been started at the lesser curvature end (5) First sero-muscular suture completed (6) An incision has been made in the stomach wall down to the mucosa, prominent vessels have been controlled by underrunning them with fine catgut.

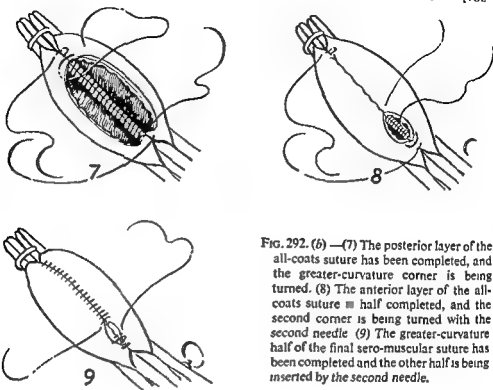


FIG. 292. (b) —(7) The posterior layer of the all-coats suture has been completed, and the greater-curvature corner is being turned. (8) The anterior layer of the all-coats suture is half completed, and the second corner is being turned with the second needle. (9) The greater-curvature half of the final sero-muscular suture has been completed and the other half is being inserted by the second needle.

Allis's forceps

long, extending backwards from within $\frac{1}{4}$ inch of the artery. The posterior wall of the stomach is pulled through this slit, and the lowest point of the greater curvature is selected and marked with Allis's forceps. A second pair of forceps is put on the posterior wall near the lesser curve where a line drawn from the first forceps at right angles to the long-axis of the stomach would strike it. A pouch of stomach is drawn up between the two forceps and grasped in the blades of one pair of a set of Lane's twin gastric clamps; the forceps are then removed. The duodeno-jejunal flexure is identified, and, starting 4 inches beyond it, a loop of jejunum 4 inches long is grasped in the second pair of clamps. The transverse colon and omentum are returned to the abdomen, leaving the gastric and jejunal loops and the clamps holding them on the surface. A strip of gauze soaked in 1:1,000 flavine solution is laid between the loops, which are then approximated—proximal end of jejunal loop to lesser curve of stomach—by locking the twin clamps together. Full-size flavine swabs are laid round and under the clamps and over their handles.

The form of anastomosis recommended is a two-layer suture, using No. 00 catgut with an atraumatic needle at each end of both sutures, each layer being reinforced by stay sutures at the ends and in the middle. Two needles are used, so that each corner is turned independently and the two sutures meet in the middle of the return layer, a device whereby the inevitable inequality that appears when one needle is used all the way round, and the ill-fitting corner where the suture ends, are avoided. A stay suture is placed uniting the stomach and jejunum at the greater-curvature extremity of the proposed stoma, another is placed at the middle, and both are held in forceps. The first continuous sero-muscular suture is then started at the lesser-curvature extremity, knotted in the middle, and one needle is laid on the towel. With the

second needle the two viscera are united by a continuous Lembert stitch, each stay suture being knotted in and cut short as it is reached. After knotting to the second stay suture, the continuous suture is taken once again through stomach and jejunum (where it will be the first stitch of the sero-muscular return layer), and the needle is laid on the towel.

The outer coats of the stomach are now divided with a knife $\frac{1}{4}$ inch away from and parallel to the sero-muscular suture, and for a length $\frac{1}{4}$ inch short of that of the first layer. Any large vessels in the submucous coat are under-run with \equiv stitch. The mucous coat is then opened throughout the length of the opening in the outer coats and the surface of the mucosa is wiped clean with small swabs, these being thrown away when they are soiled. The jejunal loop is opened with scissors for a similar length and at a similar distance from the first layer of sutures. The second suture, which includes all coats of both viscera, is inserted in the same way as the first; that is, stay sutures are put in through all coats at the greater-curvature corner and in the middle of the posterior layer, and the second double-needle suture is passed through all coats at the lesser-curvature corner and tied at its middle. From this point the suture is carried through all coats in a continuous over-and-over stitch, the stitches being placed about 6 to the inch, each held taut by an assistant till the next is through, and then laid down at right angles to the opening as the gut \equiv pulled tight. The stay sutures are knotted and cut short. The corners are turned in the same way. At the greater-curvature end the needle is passed out through stomach, in through jejunum, and pulled along the line of the stoma while a finger is pressed on the suture line. Each stitch repeats the first—out from stomach, in to jejunum, pull with the loop on the mucosa, and press on the outside. In this way a continuous haemostatic suture with full inversion is attained. At the lesser-curvature end the reverse is done, the needle is passed out through jejunum and in through stomach, but the pull on the mucosa and the pressure on the outside are the same. Finally the stitch coming out through the jejunum arrives opposite one coming out through the stomach and the two are tied together. The two needles on the ends of the sero-muscular suture are now used to infold the corners independently, and a sero-muscular suture is continued from each end, the sewing being done in opposite directions, till the two sutures meet and are tied together.

The clamps are taken off, the flavine swabs are removed and fresh towels and instruments are brought into use. The transverse colon is once more brought out of the abdomen and held up, and the edges of the slit in the transverse mesocolon are tacked by a series of interrupted sutures to the posterior wall of the stomach about $\frac{1}{4}$ inch away from the anastomosis. The transverse colon and stoma are returned to the abdomen, the omentum is laid back in place and the incision is closed.

(ii) *Finney's pyloroplasty*.—The control of haemorrhage and of soiling in a pyloroplasty is not so complete as it is in a gastro-jejunostomy, and the operation is technically more difficult. The chief advantage of the method is that it overcomes pyloric stenosis without encroaching on the infra- Useful when gastro-jejunostomy contra-indicated
except that of the pylorus. Finney's operation is possible when multiple adhesions from previous operations would make gastro-jejunostomy very difficult

The second part of the duodenum is freed by incision of the peritoneum to its outer side, and mobilized by blunt dissection (Fig. 293), till it can be laid without tension alongside the pyloric antrum and canal. Owing to the thickness of the pyloric musculature, a three-layer suture is used for the posterior

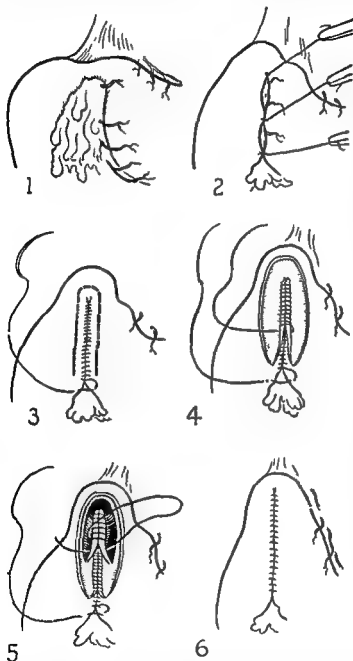


FIG. 293.—Finney's pyloroplasty. (1) Pyloric stenosis due to a cicatrizing duodenal ulcer. (2) The second part of the duodenum has been mobilized so that it can lie parallel to the pyloric end of the greater curvature of the stomach without tension; duodenum and stomach have been approximated by 3 stay sutures. (3) A sero-muscular suture has been inserted and tied to the stay-sutures; the site of the proposed opening is indicated by a dotted line. (4) The opening has been made through the serous and muscular coats only; a continuous suture is being inserted to unite the cut edges. (5) The upper part of the stoma has been opened and a continuous all-coats suture has been started. (6) Operation completed.

part of the anastomosis. One stay suture is placed on the inferior aspect of the duodenum at the site of stenosis, another joining the greater curve of the stomach and the medial border of the second part of the duodenum 2 inches from this site, and a third half-way between the two. While the two end stay sutures are held taut, a continuous sero-muscular suture is started at the first and continued down to the lowest one, where it is knotted; the stay sutures

are cut short, and the needle is laid aside. With a knife the sero-muscular layers of the pyloric antrum are divided, the cut starting $\frac{1}{2}$ inch distal to the end of the sero-muscular suture, passing up parallel to it, curving in a U across the strictured duodenum and back down the second part, parallel to itself. A second suture is started in the trough of this U-shaped cut, and continued down, uniting the inner edge of the cut duodenal wall to the outer edge of the cut pyloric wall as far as the end of the limbs of the U, where it is knotted and left long. The lumen of the duodenum is now opened at the stricture and the cut is extended $\frac{1}{2}$ inch into each limb of the U. A third all-coats suture is started at the U and continued down, the cuts in the mucous membrane being extended as the suture proceeds, till the latter reaches the ends of the two cuts, where it is tied to the long end of the previous suture which is then cut; it is then made to turn the corner, as in gastro-jejunostomy, by bringing the needle out through all coats of the stomach, in through all coats of the duodenum, pulling on the end in the lumen and pressing outside. It is continued as an all-coats inverting stitch till it comes once more to the U, where it completes the opening and is knotted. The needle belonging to the first suture is taken up and continued as a sero-muscular suture bringing the coats of stomach and duodenum together and ending at the upper border of the duodenum at the point of stenosis.

The classical Finney's operation cannot be performed if the stenosis at the pylorus is complete or if this area is surrounded by dense adhesions. In such cases a lateral anastomosis may be performed between the pyloric canal and the second part of the duodenum after free mobilization of the latter. *Finney's operation contra-indicated*

(b) Short-circuiting operations for hour-glass stomach

The simplest operation that can be undertaken to circumvent the stenosis in hour-glass stomach is gastro-gastrostomy, the proximal and distal pouches being joined by a wide anastomosis performed in a manner identical to that described for gastro-jejunostomy. An alternative operation is gastro-jejunostomy to the proximal pouch. *Gastro-gastrostomy*

If there is pyloric stenosis in addition to a mid-gastric stricture, the distal as well as the proximal pouch must be drained. The alternatives that may be employed are gastro-gastrostomy combined with Finney's pyloroplasty, gastro-jejunostomy to the proximal pouch and Finney's operation at the pylorus, or double gastro-jejunostomy, a separate loop of jejunum being anastomosed to each pouch. *Alternative procedures*

On the whole these operations are out of favour, and there is a tendency to look on gastrectomy as the operation to be performed. Wasting and dehydration can be overcome by careful preparation, the ulcer is usually not active and the acid is low, so that a moderate removal of stomach, which is all that is required, is as easy as the somewhat complicated expedients mentioned above. If the proximal pouch is high and small, gastrectomy is easier than either gastro-gastrostomy or gastro-jejunostomy, since, by reason of the ligation of the left gastric artery and the excision of the scarred lesser curve, it gives a stoma that is low and free instead of high and fixed. *Gastrectomy usually preferable*

(3) Haemorrhage

Haemorrhage from a peptic ulcer is seldom an emergency, and should be treated as such only in the small group mentioned on page 508 in which the

*Absolute rest
and trans-
fusion*

chronicity of the ulcer, its site astride a large artery, and the age of the patient, all render secure arrest of the haemorrhage by natural means unlikely. In other cases it is preferable to encourage the arrest of haemorrhage by absolute rest and transfusion, and to perform gastrectomy, the only operation that can definitely effect removal or permanent healing of the ulcer whether gastric or duodenal, as soon as the patient is fit to stand operation and before another haemorrhage occurs. An interval of 6 weeks may be looked on as satisfactory.

*Cross-
matching of
blood*

*Mode of
administration*

When an operation is necessary during active haemorrhage, it should be undertaken if possible before 48 hours have elapsed. Local anaesthesia, that is, abdominal field block followed by splanchnic and mesenteric block, is ideal if the surgeon is familiar with the technique; otherwise gas and oxygen or Trilene, reinforced by local infiltration of the abdominal wall, should be used. The patient's blood should be grouped, and all the blood to be used should be cross-matched against his serum. Six bottles of blood should be ready for immediate use, and at least 4 more should be in reserve. The first pint or two should be run in rapidly, that is, at the rate of 1 pint in 15 or 20 minutes, till the systolic blood-pressure has been raised to 100 millimetres Hg. Thereafter the rate should be suited to the condition of the patient, the amount of blood lost at the operation, and the appearance of the tissues.

*Chronic
lesser-curve
ulcer*

The first step, after opening the abdomen, is to confirm that the blood is in fact coming from a chronic peptic ulcer. If an ulcer is not felt in stomach or duodenum, search should be made for a Meckel's diverticulum containing a bleeding ulcer. If a visible or palpable lesion cannot be found to account for the haemorrhage, the abdomen should be closed rapidly and the patient should be returned to bed.

*Chronic
duodenal
ulcer*

When a chronic ulcer of the lesser curve of the stomach is found, gastrectomy, which includes removal of the ulcer, should be done unless the condition of the patient is such as to make a rapid termination of the operation imperative; with a good anaesthetist and a good blood bank such a crisis should not occur. Bleeding can be stopped, temporarily at any rate, by under-running the main vessels of the lesser curve on each side of the ulcer with silk or stout catgut, but this method should be used only as a last resort. The ulcer exposed to the gastric juices, and damaged by the handling and the sutures, is likely to spread or open the vessel again at some fresh point.

*Treatment by
gastrectomy*

When a chronic duodenal ulcer is found, the need to remove the ulcer or to attack the bleeding point directly is not absolute. A duodenal ulcer will heal from the time that the acid gastric efflux is diverted from it and will remain healed permanently. If, therefore, the ulcer is not bleeding when the duodenum is inspected, it is unwise to risk fresh haemorrhage by attempting to dissect round it or by handling it unnecessarily. Gastrectomy should be proceeded with, the distal line of division being proximal to the ulcer. Gastro-jejunostomy has been advocated in such an emergency because of its simplicity, but it does not ensure that complete sequestration of the

*Ulcer bleeding
at laparotomy*

colour of the duodenum, a direct attack must be made on the bleeding point. The alternatives that may be considered are ligation of the gastro-duodenal artery, excision of the ulcer or transduodenal approach to the ulcer bed. A fourth alternative frequently recommended, that of infolding the anterior wall

of the duodenum by sutures to form a pad which compresses the bleeding point posteriorly, is quite useless; it takes far more pressure to stop an artery than can be applied by such indirect means.

The first two alternatives are not really distinct, for the gastro-duodenal artery is so involved in the scar and oedema surrounding the ulcer bed, that any attempt to isolate it is almost bound to involve opening the ulcer itself. The choice that must be made is between excision of the ulcer and arrest of the bleeding points by suture as they lie in the ulcer bed; it will be decided almost entirely by the probable size of the ulcer crater, as estimated by palpation from without and through the duodenal wall. Some bleeding duodenal ulcers, as seen at necropsy, are an inch and a half in diameter, eroding deeply into the pancreas, involving the common duct and even extending beyond it into the second part of the duodenum. The surgeon who starts to excise one of these ulcers has done more harm than good. He is forced to stop half-way through the procedure, and to retreat from the scene of his foolhardy adventure as best he can, by suturing the cut anterior wall of the duodenum to the base of the ulcer, infolding further duodenal wall by more stitches drawing it to the edge of the crater, reinforcing the suture line with omentum, leaving in a drain and hoping for the best.

Choice of procedure

If the ulcer can be excised with speed and safety, excision is the best procedure. If not, the anterior wall of the pyloric canal and duodenum should be opened by a 3-inch incision, extending for 2 inches into the stomach and 1 inch into the duodenum. Bleeding points in this incision are rapidly seized with toothed haemostats, and these are used to hold the margin apart while a ball swab is pushed firmly into the ulcer crater. Free blood is mopped out of the lumen, and when the swab is removed the bleeding points (there are usually two) will be seen. A silk suture on a small curved needle is passed deeply through the tissues round each of these points (care being taken to avoid the common duct), and tied firmly. The duodenum is then divided just beyond the pylorus by a circular cut joining the vertical one, and closed by three layers of infolding sutures. The operation is completed by a radical gastrectomy.

Excision preferable, if possible

Radical gastrectomy

The criticisms directed against suture of the bleeding points in gastric ulcer do not apply to the same procedure when it is used in cases of duodenal ulcer. The gastric ulcer remains exposed to gastric juices and will enlarge; the duodenal ulcer is removed permanently from any contact with gastric juices, and will heal.

14. OPERATIONS FOR REMOVAL OF THE ULCER

(1) Gastric ulcer

When a gastric ulcer has reached a stage of chronicity demanding surgery, it should, if possible, be removed; even when healed there remains a scarred area, pulled on by movement, and exposed to gastric juice. True gastric ulcers very seldom become malignant, but ulcers appearing for the first time after the age of 40 are often slow cancers masquerading as peptic ulcers. All ulcers of the stomach appearing late in life and giving a history that departs in any way from the usual picture should be viewed with grave suspicion, even though the appetite remains, the acid curve is high, and there is some

Malignancy rare in gastric ulcers

improvement with rest; they should be subjected to operation unless they are healed, or nearly so, after 4 weeks of treatment.

Local operations not now favoured

Local operations for the removal of gastric ulcer, such as Balfour's cautery resection, wedge resection and sleeve resection, any of which might be done alone or combined with a gastro-jejunostomy, were formerly favoured by many surgeons. They have been abandoned, and resection of the ulcer is now performed as part of the operation of gastrectomy. The part of the stomach wall which is involved in the ulcer is removed, including the base of the ulcer—if this can be done without a difficult dissection; if, however, the ulcer erodes pancreas or liver, the base is left behind.

Irremovable ulcers

Some gastric ulcers are so large, so fixed, or encroach so closely on the cardiac orifice, that they are irremovable or barely removable. Many of these are transferred from the irremovable to the removable group after 2 weeks' intensive treatment with the "Belsen drip", but some remain that cannot be removed by the standard technique of subtotal gastrectomy, at any rate in the patient under consideration. For these two devices may be considered.

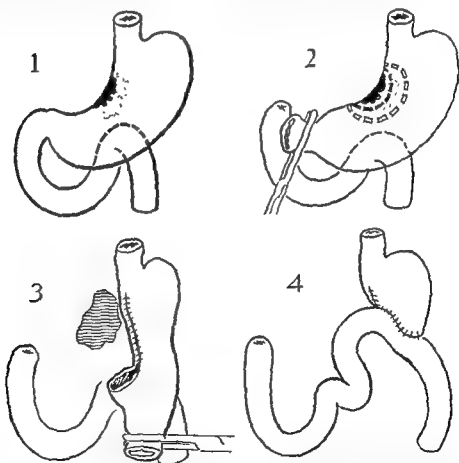


FIG. 294.—Pauchet's manoeuvre (1) Large gastric ulcer, the part visible from in front is indicated in black, the posterior part (eroding the pancreas) is shaded (2) The duodenum indicated in black, the posterior part of the ulcer eroding the pancreas (3) The stomach and duodenum with the ulcer and pancreas (4) The stomach and duodenum with the ulcer and pancreas, illustrating the final result of the manoeuvre. The base of the ulcer is seen with the pancreas. The operation is completed.

(a) *Pauchet's manoeuvre* (Fig. 294)

The greater curvature having been freed, and the duodenum having been divided, the stomach is drawn strongly downwards, an opening is made below the ulcer, and the stomach wall is trimmed away with scissors close to the edge, leaving the crater behind. More of the anterior wall can usually be preserved than of the posterior wall, and the hole that remains, converted by traction into a slit, is closed by two layers of interrupted sutures. The upper part of the stomach now appears as a tube, mostly made from the fundus, and this is anastomosed to the jejunum, the remainder of the stomach being cut away.

Closure by interrupted sutures

(b) *Gastrectomy distal to the ulcer*

When the position or fixity of the ulcer, or the condition of the patient, contra-indicates Pauchet's manoeuvre, the stomach may be divided distal to the lesion and joined to the jejunum by the Polya method. In many old people this seemingly inadequate operation is entirely successful; it differs from gastro-jejunostomy in that it removes the pyloric mucous membrane from which the stimulus to late acid secretion is derived.

Polya's method

(2) *Duodenal ulcer*

To cure a duodenal ulcer it is unnecessary to remove the ulcer; all that is required is that the gastric juice should be excluded from it. Scarring is of no importance, for after gastrectomy the duodenal stump has no function.

When the duodenum can safely be mobilized beyond the ulcer, so that after division a cuff, at least $\frac{3}{4}$ inch deep, of healthy duodenal wall remains for infolding, the ulcer should be excised; when it cannot it should be left alone. The decision must be made at an early stage of the operation, before the blood supply of the pyloric end of the stomach has been jeopardized, and there are occasions when the surgeon finds he cannot, after all, get beyond the ulcer, and must make the best job he can of a difficult closure.

Decision to excise the ulcer

(a) *Duodenal division beyond the ulcer*

The first part of the duodenum is free from vessels on its right (free) and left (pancreatic) surfaces, and receives its blood supply on its upper and lower borders. Working carefully along these borders, dividing all strands between ligatures and keeping to the plane of the duodenal wall which is maintained even through scar tissue, the surgeon can usually get beyond the ulcer. The duodenum is crushed with a Payr clamp at the level selected for division, seized with a Schoemaker clamp in the crushed strip, and cut across above it. Two methods of closure are recommended (Fig. 295).

Two methods of closure

(i) If the duodenum is narrow and its wall soft, an over-and-over stitch is run through the wall just below the clamp, which is then removed. The stitch is pulled tight, the duodenum is puckered by pushing the two ends together, and the ends of the suture are tied, closing the cut end in a tight bunch. The tied suture is held while a purse-string suture is put in the duodenal wall $\frac{1}{2}$ inch beyond it; it is then cut short, and the stump is invaginated. A second purse-string suture may be used to reinforce the first.

(ii) If the duodenum is wide, or its walls are oedematous, a running Halsted mattress suture is put into the duodenal wall. When the clamp is removed and

Halsted mattress suture

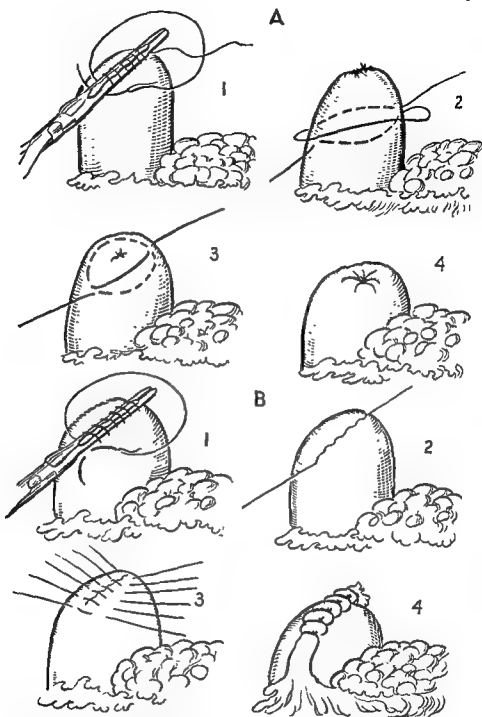


FIG. 295.—Closure of the duodenal stump

A. By over-and-over and purse-string sutures. (1) Continuous suture over a Schoemaker clamp. (2) The continuous suture is bunched and tied, a Lane purse-string suture (from 11 o'clock to 12 o'clock on the left wall, then from 6 to 12 on the right wall) is inserted $\frac{1}{2}$ inch beyond it (3) The purse-string suture is being tightened and the stump buried (4) Closure completed.

B: By continuous Halsted mattress suture, followed by interrupted Czerny-Lambert sutures and reinforced by a pedicled omental flap (1) Continuous Halsted suture over a Schoemaker clamp. (2) Clamp removed, suture drawn tight. (3) Row of Czerny-Lambert sutures inserted. (4) Sutures tied, and tied again over a pedicled strip of omentum.

this suture is pulled tight, the cut edge of the duodenum is inverted in a straight line. Six or eight interrupted sutures are then used to invaginate this line further.

The duodenal stump should not be sutured to the pancreas if this can be avoided. If doubt is felt about the security of closure by either method, the last sutures should be left long and tied again over a pedicled flap of omental fat.

(b) *Duodenal division through the ulcer*

This division should not be done intentionally; it may be unavoidable when the ulcer proves unexpectedly large, or when it breaks open during examination. The ulcers that cannot be removed safely are usually situated postero-medially.

Difficulty of removing postero-medial ulcers

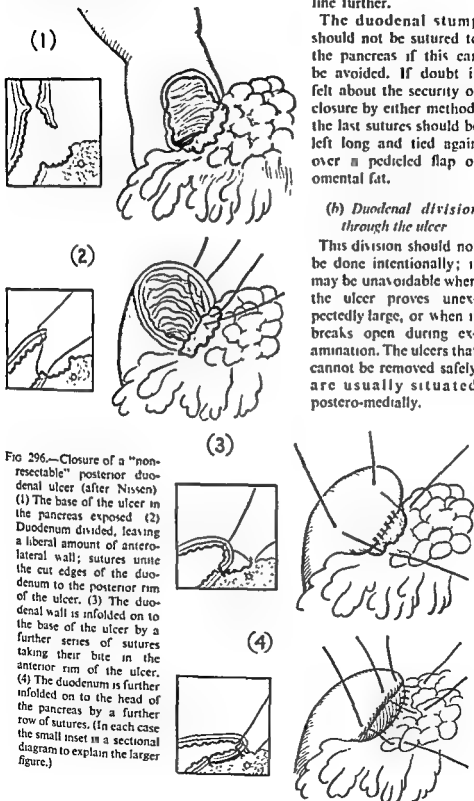


FIG 296.—Closure of a "non-resectable" posterior duodenal ulcer (after Nissen). (1) The base of the ulcer in the pancreas exposed. (2) Duodenum divided, leaving a liberal amount of antero-lateral wall; sutures unite the cut edges of the duodenum to the posterior rim of the ulcer. (3) The duodenal wall is folded on to the base of the ulcer by a further series of sutures taking their bite in the anterior rim of the ulcer. (4) The duodenum is further folded on to the head of the pancreas by a further row of sutures. (In each case the small inset in a sectional diagram to explain the larger figure.)

Many devices have been described; in an emergency the surgeon must often invent his own. The key to safety, once the ulcer has been opened, is to preserve as much of the remaining duodenum as possible; this, in most cases, consists of the antero-lateral wall. When the ulcer has been dissected as far as safety will allow, this healthy wall is trimmed in a broad flap extending nearly to the pylorus, which is joined by interrupted sutures to the posterior margin or fibrous base of the ulcer. A further row of interrupted sutures, taking their bite in the anterior margin of the ulcer, infolds this wall on to the base of the ulcer, and a third row brings the first two on to the head of the pancreas (Fig. 296).

(c) *Closure proximal to the ulcer*

It is sometimes possible to divide and infold the duodenum proximal to the ulcer, but usually this device is prohibited by the narrowness and rigidity of the part into which the closed end would have to be infolded. Pre-pyloric section of the sero-muscular coats with removal of the mucous membrane down to the pylorus, the operation recommended by Finsterer (1923) and by Bancroft (1932), is usually the safest procedure (Fig. 297).

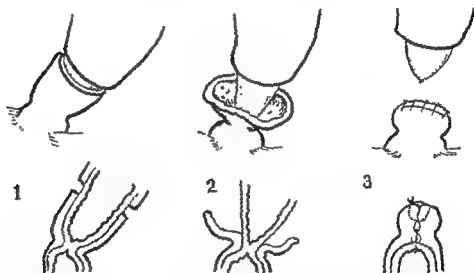


FIG 297 —Bancroft's operation. (1) The wall of the stomach has been divided in the pyloric region by a circular cut going down to the submucous layer (2) The mucous layer has been cleared down to the pylorus; a number of vessels have been tied in the submucous layer. (3) The mucous membrane has been divided and sutured at the pylorus, reinforced by 2 purse-string sutures through the ring of muscle above it, the sero-muscular coats of the gastric stump have been approximated.

*Pre-pyloric
section of
sero-muscular
coats*

The gastric vessels are ligated on both curvatures to within an inch of the pylorus. The coats of the stomach down to the submucous layer are then divided at this level with a knife, and the cut edge is picked up at four points by Allis's forceps. The mucous coat is then dissected clear down to the pylorus by pushing in the submucous layer with small swabs, dividing and tying vessels that cross this layer as they are encountered. The mucous coat is firmly adherent at the pylorus, and usually tears across at this level, a matter of no importance, for the hole can be closed by a small purse-string suture of

catgut, and reinforced by two further purse-string sutures closing the muscular ring above it. The remaining sero-muscular coats are then trimmed with an inward slope so that they can be approximated, with a series of catgut sutures.

(d) *Two-stage duodenal excision*

The operation of gastric exclusion, that is, division and closure of the stomach proximal to the pylorus without removal of the mucous membrane, is a major surgical crime. It leads to stomal ulcer with all the certainty of a reliable experiment. It may, however, be used as a temporary measure when the pylorus and duodenum are found to be widely involved in oedema and scar tissue. The second operation should be done not more than 4 weeks later, as jejunal ulcers have been known to perforate within 5 weeks of the exclusion operation that caused them. At this second operation all the oedema and much of the scarring will be found to have disappeared and the pyloric stump and first inch of the duodenum can be removed without great difficulty.

Gastric exclusion condemned

Gastric exclusion as a temporary measure

15. RADICAL OPERATIONS FOR THE PREVENTION OF FUTURE ULCERATION

For practical purposes, the only sure method by which further ulceration can be prevented is by a radical reduction in the level of acid secretion.

Reduction in level of acid secretion

Acid is secreted by the mucous membrane lining the fundus and body of the stomach, but the amount produced in the body of the stomach where the lining is thick and folded, exceeds that produced in the fundus where it is thin and smooth. Acid is secreted in response to two mechanisms—nervous impulses transmitted by the vagi, and the secretory hormone which is liberated in the mucous membrane of the pyloric antrum (and to a very much lesser extent in that of the duodenum and upper small intestine) when it is brought into contact with the products of gastric digestion, and which acts through the blood stream. Surgeons have attempted to reduce the acid level by adopting the following measures:

Mechanisms concerned in acid secretion

- (1) Neutralizing the acid, leaving its secretion unchanged.
- (2) Reducing the amount of secreting surface.
- (3) Reducing the secretory power of the surface, without reducing its area.
- (4) Cutting off vagal secretory stimuli.
- (5) Cutting off the hormonal stimulus to secretion.

(1) Operations designed to reduce the acid level without gastric resection

(a) *Short-circuiting operations*

Gastro-jejunostomy, gastro-duodenostomy and pyloroplasty of various types have been advocated as a means of reducing the acid level in the stomach, in the belief that the normal method of reduction is by neutralization. It was held that at the end of gastric digestion the pylorus relaxed, allowing the alkaline juices from the duodenum to flow back into the stomach. This conception is not supported by examination of the normal stomach on the screen during the course of a barium meal, or by the results of fractional test meals carried out with healthy individuals, in which it is uncommon to find bile in any specimen and quite exceptional to find more than a trace.

Theory of neutralization refuted

Gastric digestion is brought to an end, not by neutralization, but by the cessation of gastric secretion and the emptying of the stomach. The acid level drops immediately after any short-circuiting operation as it does after any operation that involves handling the stomach, but in a few months it returns to its pre-operative level or higher. The results of these operations, therefore, when they are performed to cure the ulcer diathesis rather than to overcome old-standing stenosis in patients past middle age, are unsatisfactory. Gastro-duodenostomy and pyloroplasty are even less suitable than is gastro-jejunostomy because the resulting failure is more difficult to put right.

(b) *Vaso-ligation*

Wilson Hey (1947) and Somervell (1945) have recommended ligation of the majority of the vessels of the stomach, claiming that the secretory power of the gastric mucosa is thereby permanently reduced. The operation has found little favour with other surgeons and the results of it have been found, on the whole, to be unsatisfactory.

(c) *Vagotomy*

Division of the vagi to lessen gastric pain has been practised sporadically for 40 years. As a means of reducing the acid level in the stomach it has been propounded chiefly by Dragstedt (1945), who showed that following supra-diaphragmatic section of the vagi in the dog, both the quantity and the acid level of the gastric secretion are greatly reduced.

Dragstedt's first operations in men were done by the transthoracic approach, the two vagi being picked up where they lie on the oesophagus in the posterior mediastinum, divided at or below the hiatus in the diaphragm, and their proximal ends stitched up to the pleura to prevent regeneration.

The majority of surgeons, including Dragstedt himself, tend to abandon the transthoracic operation, because it does not allow inspection of the ulcer or the performance of a short-circuiting operation, which is often desirable, and, further, because they consider that the operation from below is as complete. Dragstedt refers to this low operation as transabdominal rather than subdiaphragmatic, because the nerves are divided at a point at least 2 inches above the diaphragm.

The abdominal oesophagus is exposed by drawing the stomach strongly downwards and retracting the left lobe of the liver forwards and to the right, after division, if necessary, of the left lateral ligament. The peritoneum covering the oesophagus is divided transversely, the oesophagus is isolated by finger dissection, and a rubber tube is passed round it for traction. By pulling and gentle gauze dissection, 2 or 3 inches of the thoracic oesophagus can easily be brought down through the hiatus. Dragstedt has shown that in 80 per cent of cases the vagi enter the abdomen as single trunks, the left in front, the right behind the oesophagus, and both of them to the right of its central axis (Dragstedt and his colleagues, 1947) (Fig. 298).

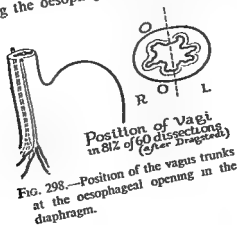


FIG. 298.—Position of the vagus trunks at the oesophageal opening in the diaphragm.

Reduction in secretory power of gastric mucosa

Supra-diaphragmatic section of the vagi

Transthoracic replaced by transabdominal approach

The left vagus can be pulled down. It is picked up where it divides. to or beyond the right vagus, similar extent. oesophagus is. Vagotomy is important in the do not abolish

The left vagus can be felt as a tight bow-string when the oesophagus is pulled down. It is picked up on a small hook, and stripped down to the point at which it divides. It is cut here, and the upper end is cleared by the hook up to or beyond the level of oesophageal clearance, where it is cut across. The right vagus, similarly taut, is hooked round with the finger and resected to a similar extent. A search is made by traction, for any uncut branches. The oesophagus is allowed to retract, and the abdomen is closed.

Vagotomy abolishes the vagal secretion of acid, the all-important mechanism in the dog, but one that is not so dominating in man, and possibly less important in the ulcer patient than is the hormonal mechanism which it does not abolish (Fig. 299). It diminishes peristalsis and prolongs emptying time;

*Abolition of
vagal secretion
of acid*

GUY'S HOSPITAL

FRACTIONAL TEST-MEAL

Name of Patient

Physician or Surgeon

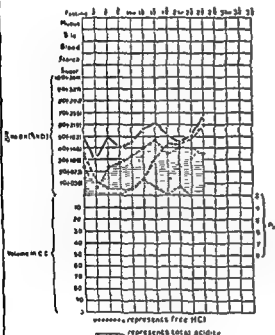
Ward

Bed

Date

REMARKS

Minutes after start of meal



OCCULT BLOOD IN STOOLS

Date	Occult Test	Haemoglobin	And Hematin	Notes

Fig. 299.—Duodenal ulcer Effects of vagal section on acid secretion. Curves of fractional test-meals obtained before and after vagotomy are superimposed. Continuous line represents free HCl on April 4, 1947, dotted line represents free HCl on October 16, 1947.

it relieves pain, the origin of which is usually in the spasmodic contractions of the muscular coat; and it allows the ulcer to heal.

The wave of enthusiasm that greeted vagotomy has moderated, and a reassessment is in process. The reduction of acid achieved at the beginning of the meal, when it is relatively harmless. The abolition of muscular contraction brings distension and many of the symptoms of stenosis, so that some surgeons consider it necessary to do a short-circuiting operation at the time of

vagotomy. These muscular contractions return later, bringing back with them the pain and, in a proportion of cases, the ulcer recurs.

Reasons for unsatisfactory results

The unsatisfactory results of vagotomy are due partly to the fact that it is such an easy operation and partly that it is being done by surgeons who have not the skill for radical surgery or the experience to allow of their making clinical decisions. It is not as yet established as the first line of treatment of the ulcer diathesis, but it provides a satisfactory means of treating duodenal ulcers that have proved resistant to treatment yet have not produced any fixed deformity, and of dealing with pain or recurrent ulceration following high gastrectomy.

(2) Gastrectomy

Definitions of Billroth I and II operations

In order to avoid the eponymity that envelops the literature of gastric surgery it is well to class all resections of the stomach as modifications of the Billroth I or Billroth II operation. The Billroth I operation is gastrectomy followed by gastro-duodenostomy; the Billroth II operation is gastrectomy followed by gastro-jejunostomy. The modern descendants of both operations are satisfactory, provided that they fulfil certain requirements. These requirements are enumerated below.

(a) The whole of the pyloric antrum or its mucous membrane must be removed in order to abolish the action of the antral hormone.

(b) The body of the stomach containing the major part of the acid-secreting mucous membrane must be removed.

(c) The lesser curve, or at any rate its mesentery, must be divided close to the cardia, in order to cut the vagal branches, most of which are bunched here, and to free the stomach from the anchoring effect of the left gastric artery.

(d) The cut surface of the stomach must be closed in part so that the diameter of the stoma is no larger than that of the intestine into which it empties. The curtailed stomach should not be made into a funnel, shooting each mouthful straight into the jejunum, but should retain the meal for a period of gastric digestion.

(e) After leaving the stomach the meal should pass in an onward direction only.

Anastomosis of the whole width of the divided stomach to the jejunum, whether the stoma be antecolic or retrocolic, iso-peristaltic or anti-peristaltic, is an unsatisfactory operation that should be reserved for cases of special difficulty or as a palliative measure in cancer surgery. Partial closure of the stomach is made much easier by the use of the Friedrich-Petz sewing clamp (Fig. 300).

(a) Freeing the stomach

The early steps of gastrectomy, which involve freeing the stomach and controlling its blood supply, are the same whatever stoma is used, and are trodden in the same order by most surgeons; that is, first greater curve, then

The tying of the vessels

...ve the gastro-epiploic
...ply to the great omen-
...warden" unpaired.

The second course leaves the omentum a purple ridge; this is desirable in

gastrectomy for cancer when the lymphatic glands accompanying the main vessels must be removed, and it is easier in gastrectomy for ulcer since it saves time. The choice is more a matter of taste than of practical importance. The surgeon-artist, who wishes to damage nothing but the structure which he

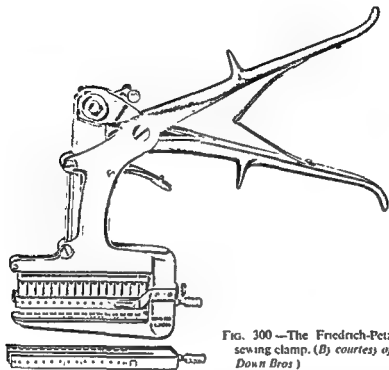


FIG. 300.—The Friedrich-Petz sewing clamp. (By courtesy of Down Bros.)

intends to remove, will choose the first course just as he will approach the thyroid gland by retracting the infrahyoid muscles to either side, the surgeon-artisan who wants to do a sound job quickly and get on to the next, will tie omentum in bunches and cut across the strap muscles, claiming with some justice that in neither case can any real harm be demonstrated afterwards.

The first vessels to be divided are those where the right and left gastro-epiploic territories meet, in this way the lesser sac is opened where it is free from adhesions. Division of vessels

The left index finger is hooked under the omentum, and the gastric branches are divided and ligated in turn. When an adequate opening has been made, it is held open, and the adhesions of transverse mesocolon to the back of the pyloric antrum are cleared by scissors and blunt dissection. The process of division and ligation is then continued to within 1 inch of the pylorus. At this point the decision must be made whether the distal line of section is to be pre-pyloric or post-pyloric—a decision the implications of which have already been discussed. After section and distal closure, the cut pyloric end is wrapped in a gauze swab and the stomach is held upwards.

With duodenal ulcer this is easy; with gastric ulcer it may be necessary first to dissect the ulcer from its bed. The main trunk of the left gastric artery is then put on the stretch by pulling the stomach out of the abdomen towards the patient's left shoulder; it is then divided between silk ligatures. The stomach is drawn down again, and the lesser curvature is cleared down to the

Pre-pyloric or post-pyloric line of section

muscle coats by dividing peritoneum, vagal branches and a few vessels that intervene. The greater curvature is cleared to a point that allows removal of at least three-quarters of the stomach.

(b) *The Billroth I operation (Fig. 301)*

The modern Billroth I operation satisfies all the criteria outlined above. It is the best form of gastrectomy for gastric ulcer, when the acid is not unduly high and the duodenum is normal. It is unsuitable if the duodenum is scarred by previous ulceration, and is unwise if it is large, since this suggests some degree of duodenal ileus.

The Petz clamp is first applied at right angles to the greater curvature for a distance of $2\frac{1}{2}$ inches, and the crushed strip is cut with scissors between the

Contra-
indications

The Petz
clamp

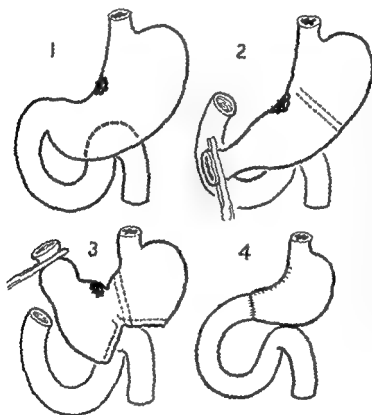


FIG. 301.—The Billroth I gastrectomy. (1) Gastric ulcer in typical situation on lesser curve. (2) Duodenum divided; Friedrich-Petz clamp applied at right angles to greater curve. (3) The first crushed strip divided; the clamp applied again from the central end of the first cut to a point higher on the lesser curve. (4) Operation completed.

two rows of clips. The clamp is applied again at right angles to the central end of the first cut, extending to the upper part of the cleared lesser curve. The second crushed strip is cut half an inch at a time between the clips, starting at its distal end, and infolded over the clips by a continuous Lembert suture. When this suture is finished the new lesser curve thus formed is found to be of ample length to allow the distal end of the stomach, opened by cutting away the row of clips first inserted, to be anastomosed to the duodenum without tension. A single layer of interrupted sutures embracing all coats is recommended for the anastomosis, this being reinforced on the anterior aspect only by a second continuous layer. The operation is completed by re-attaching the gastro-epiploic arch to the stomach and duodenum with a few sutures.

(c) *The Billroth II operation (Fig. 302)*

The modern version of the Billroth II operation involves a valvular non-Valvular

15

becomes circular. The anastomosis may be made in front of or behind the colon, but the retrocolic anastomosis is preferred.

The Petz clamp is applied obliquely from the greater curve to a point on the lesser curve about 1 inch below the cardia. The stomach is drawn down, and an infolding suture is started close to the cardia, and continued down the proximal line of clips, the crushed strip being divided with scissors, half an inch at a time, in advance of the infolding suture. When this continuous suture reaches a point $2\frac{1}{2}$ inches from the greater curve, it is tied. A further $\frac{1}{2}$ inch of clip line is infolded by three separate sutures tied and cut short, leaving the stomach still attached by a 2-inch bridge of crushed and doubly stapled tissue.

It should be noted here that the modifications of the Billroth I and II operations described may be performed equally well without the Petz clamp. A continuous all-coats suture of catgut, or a series of interrupted sutures of catgut, thread or silk, replace the row of metal clips, the stomach being controlled proximal to the line of division with a light spring clamp.

The stomach is now held in the right-hand member of a pair of gastric clamps applied 2 inches proximal to the proposed stoma. The transverse colon is held out of the wound, the duodeno-jejunal flexure is identified and the peritoneal bands holding it to the mesocolon are divided. A hole, the breadth of two fingers, is made into the lesser sac through the bare patch thus produced, and through it the jejunum is pulled, obliterating the duodeno-jejunal angle. A loop is selected some 10 or 12 inches from the flexure, held in the left-hand gastric clamp, and approximated to the stomach, proximal end to lesser curve. The anastomosis is made in a manner identical with that described for gastro-jejunostomy, the stomach being removed after the first sero-muscular layer has been completed, by cutting through above the proximal row of clips. After removal of the clamps, the jejunum proximal to the stoma is hitched up to the infolded lesser curve by a series of 3 or 4 sutures, each of which takes a double bite of the anti-mesenteric border of the jejunum together with one each side of the gastric fold. At the conclusion the jejunum should flow in a continuous curve from the flexure up to the cardia and then down the closed stomach to the stoma. Finally the transverse colon is pulled forwards once more, the emerging jejunum is seized as it leaves the opening and drawn down till the lower end of the stoma appears below the mesocolon, and the edges of the hole are fixed to the stomach wall by two sutures

*Obliteration
of duodeno-
jejunal angle*

16. POST-OPERATIVE CARE

The patient is returned to the ward with a gastric tube, passed through the nostril, in position. As soon as he is conscious, he is given fluid feeds—up to 4 ounces every hour—of any liquid that can be recovered through the tube, that is, water, tea, fruit drinks, citrated milk or Bovril. Five minutes before the next feed, the stomach is emptied with a syringe. As soon as the amount

*The gastric
tube*

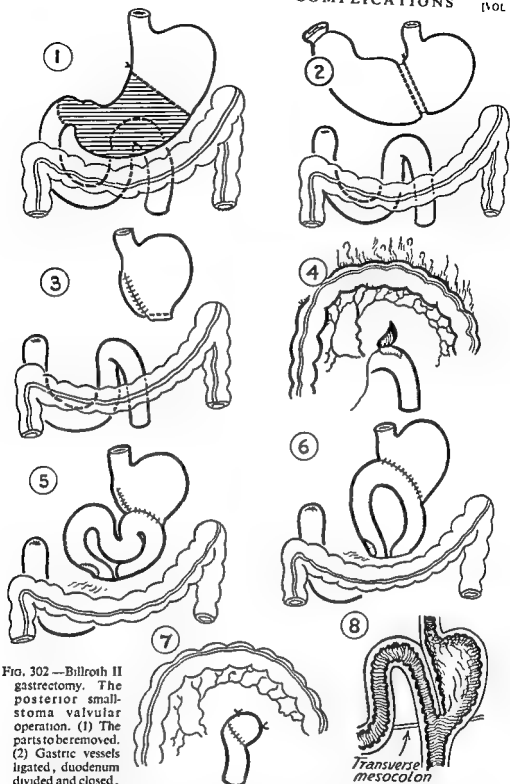


FIG. 302.—Billroth II gastrectomy. The posterior small-stoma valvular operation. (1) The parts to be removed. (2) Gastric vessels ligated, duodenum divided and closed, Friedrich-Petz clamp applied across stomach at line of intended division. (3) Upper part of line of division infolded. (4) Transverse colon turned up, attachment of duodeno-jejunal flexure to transverse mesocolon separated. (5) Long loop of jejunum pulled through

(8) Finished operation shown in section.

recovered is less than half that swallowed, aspiration is done before alternate feeds only, and ice-cream is added to the diet. When 2-hourly aspiration brings back no more than 1 ounce of fluid, the tube is withdrawn.

The routine employment of gastric suction is important, not only because it allows fluids to be given by mouth without the risk of distension, but because it gives warning of haemorrhage. The tube can usually be withdrawn after 48 hours, but occasionally it is needed for a week. During the first few days, in fact till feeds of 6 or 8 ounces are allowed, the fluid intake is supplemented by rectal salines.

*The importance
of routine
gastric
suction*

17. RESULTS OF TREATMENT

A lengthy discussion of statistics would be out of place. Personal statistics are too good to be true and massed statistics are too true to be good. Four surgeons, two working in London and two in the Midlands, have kindly given me their figures which have not yet been published.

Gastrectomy (lesion not stated)	-	-	-	-	263
Deaths	-	-	-	-	9
Mortality	-	-	-	-	3.7 per cent
Gastrectomy for duodenal ulcer	-	-	-	-	525
Deaths	-	-	-	-	10
Mortality	-	-	-	-	1.9 per cent
Gastrectomy for gastric ulcer	-	-	-	-	668
Deaths	-	-	-	-	20
Mortality	-	-	-	-	3.0 per cent
Total gastrectomies for ulcer	-	-	-	-	1,456
Total deaths	-	-	-	-	39
Mortality	-	-	-	-	2.7 per cent

Nearly half (48 per cent) of these operations were performed in two hospitals that deal with the aged and chronic sick, and the patients presented risks well above the average. The following figures would be attained by a skilful and experienced surgeon, working with average material, assisted by a good anaesthetist, and served by good theatre and ward teams.

Hospital mortality

Short-circuiting operations	-	-	-	-	11.2-20.5 per cent
Gastrectomy for ulcer	-	-	-	-	2.0-3.0 "
Secondary operations (for gastro-jejunal ulceration, etc.)	-	-	-	-	5.0-8.1 per cent

Results (among survivors)

Gastrectomy for gastric ulcer	-	-	-	-	
Satisfactory after five years	-	-	-	-	95 per cent
Improved but not symptom-free	-	-	-	-	4 "
Worse (including re-ulceration)	-	-	-	-	1 "
Gastrectomy for duodenal ulcer	-	-	-	-	
Satisfactory after five years	-	-	-	-	90 per cent
Improved but not symptom-free	-	-	-	-	8 "
Worse	-	-	-	-	2 "

Unsatisfactory sequelae

As might be expected, unsatisfactory sequelae are very often due to faulty surgery. A very honest analysis is that of Heuer (1944), who reports about 30 per cent of unsatisfactory results, including recurrent ulceration, haemorrhage and perforation, in the gastrectomies performed (88), less than half the

stomach was removed in 31, half to two-thirds in 46, and more than two-thirds in 11 only; that is, 87.5 per cent of the resections were inadequate.

The following sequelae are encountered.

(a) *Minor inconveniences*

Faintness after meals rich in sugar, and intolerance to alcohol are sometimes complained of after gastrectomy; these symptoms are due to the rapidity with which two highly absorbable foodstuffs reach a highly absorbing surface.

(b) *Dumping*

A few patients complain that they feel the meal rushing out of their stomach and filling the intestine; there may be the sense of hurry only, or an accompanying feeling of distension. Dumping is uncommon if the stoma is kept small.

(c) *Proximal loop distension*

If food enters the proximal loop in any quantity, a feeling of epigastric pain is experienced immediately after meals; this may take a couple of hours to disappear. The symptom is uncommon even after a Polya anastomosis, and unknown when a valvular stoma is made.

(d) *Recurrent ulceration*

Recurrent ulceration is practically unknown when a gastrectomy, fulfilling the five requirements outlined above (see p. 532), has been performed. After exclusion gastrectomy it occurs in from 40 to 60 per cent of cases. After a Billroth I operation has been performed in error on a patient with duodenal ileus, a duodenal ulcer may develop beyond the stoma.

BIBLIOGRAPHY AND REFERENCES

- Allen, A. W. (1944). *J. med. Ass. Ga.*, 33, 237.
 — and Welch, C. E. (1946). *Ann. Surg.*, 124, 688.
 Bancroft, F. W. (1932). *Amer. J. Surg.*, 16, 223.
 Cushing, H. (1932). *Surg. Gynec. Obstet.*, 55, 1.
 Davies, D. T., and Wilson, A. T. M. (1937). *Lancet*, 2, 1353
 — — (1939). *Ibid.*, 2, 723.
 Devine, H. B. (1925). *Surg. Gynec. Obstet.*, 40, 1.
 — (1928). *Ibid.*, 47, 239.
 Dodd, H. (1947). *Brit. med. J.*, 2, 170.
 Dragstedt, L. R. (1945). *Ann. Surg.*, 122, 973.
 — Fournier, H. J., Woodward, E. R., Tovee, B., and Harper, P. V. (1947)
Surg. Gynec. Obstet., 85, 461.
 — — — — — 17, 742.
 — — — — — 5, 5.
 — — — — — 6, 6.
 — — — — — London; Lippincott.
 — (1946). *New Engl. J. Med.*, 235, 111.
 Hey, W. H. (1947). *Brit. med. J.*, 2, 395.
 Hurst, A. F., and Stewart, M. J. (1929) *Gastric and Duodenal Ulcer*. London:
 Oxford University Press.
 — — — — — *Brit. med. J.* 1, 689.
 — — — — — 11, 2, 641

— and Marshall, S. F. (1943). *Surg. Gynec. Obstet.*, 76, 641.

Lawson, R. S. (1945). *Aust. N.Z. J. Surg.*, 14, 233.

Lewisohn, R. (1925). *Surg. Gynec. Obstet.*, 40, 70.

— (1947). *J. Mt. Sinai Hosp.*, 14, 470.

Maingot, R. (1936). In *Post-graduate Surgery*, ed. by Maingot, R., Vol. I, p. 177. London; Medical Publications.

— (1945). *Post-Grad. med. J.*, 21, 211.

Nissen, R. (1945). *Duodenal and Jejunal Peptic Ulcer*. London, Heinemann.

Ogilvie, W. H. (1935). *Brit. med. J.*, 1, 457.

— (1936). *Edinh. med. J.*, 43, 61.

— (1938). *Lancet*, 2, 235, 295.

— (1947a). *Aust. N.Z. J. Surg.*, 17, 3.

— (1947b). *Ann. R. Coll. Surg. Engl.*, 1, 37.

— (1947c). *Lancet*, 2, 377.

Robertson, H. E., and Hargis, E. H. (1925). *Med. Clin. N. Amer.*, 8, 1065.

Rosenow, E. C. (1923). *J. Infect. Dis.*, 33, 248.

Somervell, T. H. (1945). *Brit. J. Surg.*, 33, 146.

Stewart, M. J. (1923). *Brit. med. J.*, 2, 1021.

Sweeney, J. S. (1944). *Northw. Med., Seattle*, 43, 36.

Tanner, N. C. (1946). *Bristol med.-chir. J.*, 63, 16.

Theile, P. (1919). *Ergebn. inn. Med. Kinderheilk.*, 16, 302.

Walton, J., and others (1944). *Proc. R. Soc. Med.*, 38, 91.

Wolf, S., and Wolff, H. G. (1943). *Human Gastric Function*. London; Oxford University Press

Wood, W. Q. (1945). *Edinh. med. J.*, 52, 433.

[References to other titles are given under Peptic Ulcer with Complications, in the Index Volume. The subject is also dealt with under the heading of Peptic Ulcer in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, p. 504.]

PERFORATING ULCER OF THE FOOT

BY GERALD THOMAS MULLALLY, M.C., M.S., F.R.C.S.
SENIOR SURGEON, WESTMINSTER HOSPITAL; SENIOR SURGEON, HOSPITAL OF
ST. JOHN AND ST. ELIZABETH, LONDON

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1. DEFINITION

263.] Perforating ulcer is the name given to certain painless chronic ulcers of the sole of the foot which penetrate deeply into the tissues of the foot and show very little tendency to spread superficially.

2. AETIOLOGY

The cause of the condition is obscure. The freedom from pain suggests that they are in some sense "trophic" ulcers. They are found in association with tabes dorsalis, peripheral neuritis, nerve injuries and other conditions in which there is clear evidence of interference with sensory conduction. They appear also in association with arterial disease (especially arteriosclerosis), with diabetes mellitus (even without gross evidence of arterial disease or peripheral neuritis), with old poliomyelitis and, perhaps most commonly, when any of these conditions is combined with senility. They may also occur in old people (almost always males) who show no gross evidence of any disease. It is the writer's experience that examination *always* shows definite, if patchy, blunting of sensation to pin-prick on the affected sole.

Since the process is painless, the ulcer is not seen until well established, and it is always difficult to get a clear description of the initial signs and symptoms. It seems probable that the sequence of events is somewhat as follows.

The condition begins as a large corn under the heel, or under the head of the first or fifth metatarsal bone. Under the corn an adventitious bursa forms. The bursa becomes infected, but since the area is insensitive, no pain is felt and the patient continues to walk about on the foot. As the small abscess enlarges it is completely barred from the normal extension to the surface by the very thick callosity in which the corn originated. Extension, therefore, is deflected inwards through the deeper layers of the skin into the subcutaneous tissues. At this stage the process is not an acute spreading infection but one of simple ischaemic necrosis. Eventually a chronic abscess is formed which has penetrated the living skin, but is still enclosed by the thick horny overlying layer.

'Trophic' ulcers

Painless process

Corn and bursa

Chronic abscess

Gradually the horny layer gives way and the patient finds a small hole in the sole of the foot from which a thin pus exudes in small quantity. The suppuration is not due to any specific infection. The pus contains a variable sample of pathogenic and non-pathogenic organisms. In other cases the patient discovers that his sock is wet with discharge and, on investigation, finds a nail, which has been the initial cause of the trouble, projecting from the inside of his shoe.

3. MORBID ANATOMY

The ulcer is a small hole (about 0.5 centimetre in diameter) situated under the head of the first or fifth metatarsal bone, the head of the first phalanx of the great toe, the heel, or in relation to other weight-bearing bony points in deformed feet. It lies in the centre of a wide area of cornification which is up to 0.5 centimetre thick. The edge of the ulcer is thick and often fringed with sodden projections of the horny layer. These fringes overhang the actual hole in the living skin which is usually somewhat wider than the visible external

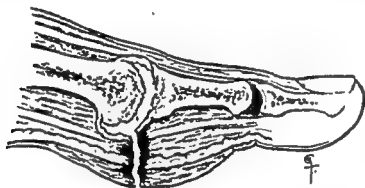


FIG. 303.—Diagrammatic illustration of a perforating ulcer extending deeply and penetrating the metatarso-phalangeal joint. Note the very thick callosity which plays so important a role in the aetiology of the disease.

ulcer. This hole is filled with pale, moist granulations so that it is not obvious that the process extends deeply into the foot. With a probe, however, a narrow track will be found going on towards the bone. The track may end blindly in the soft tissues, or may lead to a tiny patch of bare, soft bone, to an open tendon-sheath, or into the metatarso-phalangeal or interphalangeal joint (Fig. 303).

4. CLINICAL PICTURE

Usually the patient complains of a discharging place on the sole of the foot which is not painful but is uncomfortable and inconvenient. Sometimes, however, it is not the ulcer which causes him to seek advice, but swelling of the foot, and, less commonly, pain. In most cases the condition causes very little disability beyond the interference with walking and the trouble of having to wear a dressing. More serious complaints are usually the result

of complications. The whole process is very chronic and may persist over many months without important advance or regression.

5. COMPLICATIONS

The morbid anatomy suggests the likely complications.

(a) *Swelling of the foot*

Swelling of the foot is most marked on the dorsum and extending up the leg, and may be merely the result of insufficient rest, may indicate exacerbation of the local infection, or may be the chief indication of one of the more serious complications mentioned below.

(b) *Invasion of the bone*

Bone invasion is usually quite superficial, giving rise to a small area of rarefaction and softening, but may lead to gross osteomyelitis or the formation of sequestra.

(c) *Invasion of a joint*

Involvement of a joint is a common sequel, is often surprisingly undramatic, and may be overlooked unless the possibility is borne in mind. There is usually local pain in the joint which, viewed from the dorsal aspect, is swollen and may even be a little red. When the cartilage is destroyed, grating may be detected if the surfaces are rubbed together. Very occasionally pus from the joint may point on the dorsal surface on one or other side of the extensor tendon.

(d) *Involvement of tendon sheaths*

Tendon-sheath involvement is perhaps the most serious complication. It gives rise to a low-grade suppuration which is liable to spread along the tendon and so carry the infection throughout the sole of the foot, in very much the same manner as the infection which so often complicates diabetic

Spread of infection

disturbance; it is an important point in diagnosis since beyond local oedema of the foot and leg, and some general tenderness of the sole, there is very little evidence of what is going on in the interior of the sole.

6. TREATMENT

As might be expected from what is known of the aetiology of the condition, the treatment is unsatisfactory, tedious and frequently unsuccessful. Obviously, the first indication is to rest the part by keeping it elevated and by forbidding weight-bearing. The second indication is to improve the drainage. This is best achieved by paring away the overhanging horny layer with a razor or sharp scalpel. This chiropodical treatment will need to be repeated at least once a week, and perhaps more frequently, because the horny layer re-forms with great rapidity. Simple antiseptic dressings such as surgical spirit or a sulphonamide powder are better than moist dressings or iodine. Since the process is not primarily infective, penicillin and the sulphonamides have no specific value although they may cause some temporary improvement if the

Indications

PERITONEUM AND PERITONITIS

BY R. MILNES WALKER, M.S., F.R.C.S.,
PROFESSOR OF SURGERY, UNIVERSITY OF BRISTOL; SURGEON, BRISTOL ROYAL
HOSPITAL.

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1. GENERAL

(1) Importance of the peritoneum in surgery

264.] It is scarcely credible that less than a century ago the attitude of surgery to the peritoneal cavity was epitomized in the following sentence, written in 1853 by one of Lister's teachers: "These operations are all defective in one most important respect, for, as the peritoneum must, in all of them, of necessity, be wounded, an intense and frequently fatal peritonitis is the inevitable consequence." The first result of Lister's introduction of antiseptic surgery,

which made the abdominal cavity a relatively safe field for exploration, was that surgeons rushed to exploit the opportunities that it offered and a great new branch of surgery was born.

The reasons for this are not far to seek. The danger of the peritoneal cavity lies in the fact that it is the largest free space in the body, inflammatory exudate can spread rapidly from one end of it to the other, and absorption is very free from its lining serous membrane. Its advantages to the surgeon are that through one incision he can explore a large number of organs, and with very little dissection he can mobilize many of them and thus easily carry out operative procedures upon them. It is, however, only in recent years that the defensive powers of serous membranes, and particularly of the peritoneum, against bacterial invasion, have come to be fully appreciated, and that it has been recognized that this resistance is at least as great as, and probably greater than, that of other soft tissues. Given the opportunity, the peritoneum will localize infections by the formation of adhesions, in which the omentum usually plays a prominent part, and in time the natural course is for these adhesions to be absorbed again, with the restoration of the normal anatomy.

*Formation
of adhesions*

(2) Anatomy

From the surgeon's point of view the importance of the anatomy of the peritoneal cavity may be summarized as follows. Normally it is occupied by only just sufficient fluid to allow its more mobile contents to slide smoothly against each other or against the parietal peritoneum; in the mechanics of such a thin layer of fluid, surface tension must play a large part, but when the abdomen has recently been opened at operation and air has been introduced, gravity takes a larger share in the disposition of the abdominal organs and of any excess of fluid. In this case the anatomy of the posterior abdominal wall assumes some importance because, with the patient lying in the supine position, three hollows are formed in which fluid may accumulate—the pelvis, and two paravertebral gutters, one on each side. These latter lead up to the region of the liver and spleen and by a combination of surface tension and respiratory movement a flow of fluid into the potential spaces between the diaphragm and these organs is induced. There is, of course, a fourth trough in the lesser sac, but it is most unusual for infection to spread directly through the foramen of Winslow into this space.

It has been shown that particulate matter is absorbed very quickly from the peritoneal cavity by the diaphragmatic lymphatics, and clinical experience of the serious nature of infection in the upper abdomen shows that the same is true of bacterial toxins; thus the importance of avoiding spread of infection to this region has long been felt and the adoption of a semi-sitting position (Fowler's position) has been advocated and has stood the test of time. Recently, doubt has been thrown on the efficiency of this position in preventing fluid from migrating to the subphrenic spaces (Spalding, 1946), particularly during the early post-operative period when a small pneumoperitoneum is present, and many surgeons are not so strict as they formerly were in maintaining the high Fowler position during the early post-operative period. It is, perhaps, more important to maintain free respiratory movements, and the patient is usually the best judge of the position in which maximal pulmonary ventilation is obtained.

*Absorption
from
peritoneum*

*Fowler's
position*

Effect of gravity

The effect of gravity in the spread of infection has probably been overestimated, for a protective layer of adherent fibrin will form in a surprisingly short space of time and thus localize infection. Thus in the case of a perforated peptic ulcer it is not unusual, within a few hours of perforation, to find a line of such fibrin uniting the transverse colon and great omentum to the anterior abdominal wall, thus limiting the inflammation to the upper half of the abdomen.

2. ACUTE PERITONITIS

(1) Aetiology

Streptococcal and pneumococcal infection

Acute peritonitis may arise as a blood-borne infection (streptococcal or pneumococcal) but much more commonly it is the result of spread of infection from a local lesion in or adjacent to the peritoneal cavity. In such a case it will be a spreading peritonitis from the onset, but if the local defences are adequate it will become cut off from the general peritoneal cavity by adhesions and thus become a localized peritonitis. If the defence mechanisms fail, the whole of the peritoneal cavity, with the exception perhaps of the lesser sac, will become infected and such general peritonitis, therefore, carries a very high mortality rate. If the infecting organism is virulent, death may occur during the spreading stage, but when the patient has a normal resistance, and the source of infection does not continue, localization is the rule. Such localized peritonitis may clear up spontaneously or it may result in a local intraperitoneal abscess. This abscess may be anywhere in the peritoneal cavity, but there are certain common sites, depending usually upon the origin of the infection. Their order of frequency and common causes are:

Localized abscess

- (1) Right iliac fossa - - - - - appendicitis.
- (2) Pelvis - - - - - appendicitis, salpingitis, diverticulitis.
- (3) Right subhepatic - - - - - perforated duodenal ulcer, cholecystitis, appendicitis.
- (4) Right subphrenic - - - - - perforated duodenal ulcer, cholecystitis, appendicitis.
- (5) Left subphrenic - - - - - perforated gastric ulcer, acute pancreatitis.
- (6) Left iliac fossa - - - - - diverticulitis

A posterior left subphrenic abscess is associated with infection in the lesser sac.

(2) Bacteriology and pathology

Primary acute peritonitis may be associated with infection by streptococci, gonococci or pneumococci, and probably organisms reach the membrane by the blood stream, though in the female they may gain access by the Fallopian tubes without evidence of inflammation in the tubes.

In secondary peritonitis due to local spread a great variety of organisms has been found; when the primary lesion is in the alimentary canal, these are most commonly *Bacillus coli* and streptococci; from the pelvic organs the infection is often due to similar organisms, but a gonococcal peritonitis may arise as a result of infection of the Fallopian tubes. Actinomycosis does not cause diffuse peritonitis but may be the infective agent in a localized intraperitoneal abscess.

The first change seen in peritonitis is hyperaemia, the dilated blood-vessels giving a pinker colour to the membrane; shortly afterwards the surface loses its normal shiny appearance and becomes coated with a fine layer of fibrinous exudate. Fluid, which is at first serous but early becomes turbid, is also liberated into the cavity and, on the infection becoming localized, this fluid thickens to frank pus. In streptococcal infection the fluid tends to remain serous and less fibrin is deposited, whereas in pneumococcal peritonitis the reverse obtains and thick plaques of fibrin are deposited on the surface of the membrane. Sometimes, either at necropsy or in the course of operation, one may find localized abscesses containing thick pus, while other parts of the peritoneal cavity contain only thin, serous fluid.

Chemical agents may give rise to peritonitis, but in such cases secondary invasion by bacteria nearly always occurs within a short time. Examples are the gastric juice in cases of perforated peptic ulcer, bile in cases of leakage after operation on the biliary tract or, rarely, occurring spontaneously, and urine in the case of intraperitoneal rupture of the bladder. Such chemical peritonitis produces a great degree of shock, which may even be fatal, and unless treated early, secondary bacterial peritonitis supervenes within the course of a few days; only rarely does such a peritonitis become localized, spontaneously giving rise to an abscess.

Peritoneal irritation which, clinically, may mimic peritonitis, is associated with haemorrhage into the peritoneal cavity, but in such a case no inflammatory reaction is present in the serous membrane.

When *Bacillus coli* is the infecting agent, the pus has a characteristic odour, but streptococcal, pneumococcal or gonococcal infections have no smell.

(3) Chemical pathology

Patients suffering from spreading or generalized peritonitis are usually troubled by persistent vomiting; in addition their raised temperature may cause them to lose an excessive amount of moisture by the skin and respiratory tract, so that they rapidly become dehydrated and in addition lose chlorides from the stomach. This fluid loss must be made good by parenteral administration, and a watch should be kept on the urinary chlorides, since absence of chlorides from the urine indicates a serious chloride deficiency; usually these patients excrete little or no chloride in the urine and saline has to be administered freely. If vomiting is continued for some time, the plasma proteins may fall and administration of plasma may be required.

(4) Clinical features

(a) Primary acute peritonitis

This is a rare condition and, in view of its close similarity to peritonitis resulting from the spread of a local infection, caution should be exercised in making such a diagnosis. With the exception of pneumococcal peritonitis it is doubtful whether it can be recognized clinically, and the absence of evidence of a primary lesion is not sufficient to justify the diagnosis. The symptoms and signs resemble those of secondary acute peritonitis.

(b) Primary pneumococcal peritonitis

This disease is practically confined to girls under 10 years of age, the infection probably reaching the peritoneum by way of the Fallopian tubes. There

Effusion

Chemical peritonitis

Peritoneal irritation

Fluids and chlorides

Age and sex incidence

is a sudden onset with abdominal pain, vomiting, and constipation for a day or two, followed by diarrhoea; the temperature is above 100° F., the pulse is raised, and generalized abdominal rigidity and tenderness are present. A characteristic feature which may confirm the diagnosis is that the alae nasi show active movement, as in lobar pneumonia. If the diagnosis is certain, treatment by penicillin should be instituted at once; the prognosis is good, recovery usually being complete in a few days, but a residual abscess may form, most commonly in the pelvis, and this will require drainage. If there is any doubt in the diagnosis, laparotomy should be undertaken unless the child is very ill, in which case conservative measures as described for secondary acute peritonitis should be instituted. If a laparotomy is performed, the characteristic thick, odourless pus with heavy deposits of fibrin will be found; the pus should be swabbed out, the wound closed without drainage and penicillin administration commenced.

Treatment

(c) Secondary acute peritonitis

In this condition there will be manifestations of the primary lesion, the onset of peritonitis may occur with dramatic suddenness, as with a perforated peptic ulcer, and this may be the first sign of illness. In cases of acute appendicitis it is not so sudden, evidence of peritonitis being usual on the second or third day of the illness, whereas in other cases, as in acute cholecystitis or in diverticulitis of the colon, the spread to the peritoneum may be silent and show itself only when a local abscess forms.

Symptoms and signs

The symptoms and signs of the onset of peritonitis are pain, which, though localized at first to the site of the lesion, rapidly becomes more widespread; vomiting, which is a regular symptom while the peritonitis is spreading; a rise in pulse rate; and localized tenderness and rigidity. If tenderness was present as a result of the primary lesion, its area is increased. Rigidity is always found if carefully elicited, the temperature is usually raised to between 99° F. and 100° F. but it may be normal or subnormal, and is no guide to the diagnosis. Constipation is usual but diarrhoea may occur, especially in children or in cases in which the peritonitis commences in the pelvis. We have observed a few cases in which frequency of micturition has been a prominent symptom of early pelvic peritonitis, and even frank haematuria occurs, which may confuse the diagnosis; in such cases, usually associated with appendicitis, the wall of the bladder has shown inflammation spreading from the adjacent peritoneum. Cyanosis may be a prominent feature, due to restricted respiratory movement, either as a result of the rigidity or later due to the distension; when present early, the differential diagnosis from acute pancreatitis should be considered.

As peritonitis becomes generalized the classical picture of the sunken face, hiccup, distended abdomen and small rapid pulse, together with rapid wasting, makes its appearance, but the diagnosis should be made before this state is reached.

(d) Localized abscess

When this stage is reached, usually after the third day of the illness, the patient has fever, often 101°–103° F., a raised pulse rate, and very varying degrees of toxæmia; in the usual sites the abscess may be felt as a fixed,

rounded swelling which is usually tender. Unless a pelvic abscess is very large *Pelvic abscess* it will not be felt above the pubes, and rectal examination is necessary to palpate it. Here the abscess may cause symptoms as a result of irritation of neighbouring organs, for example, irritation of the rectum causing diarrhoea and the passage of mucus, or of the bladder causing increased frequency of micturition and, rarely, slight haematuria. Subphrenic abscesses are the most difficult to detect but their recognition is most important as they cause the most severe degree of toxæmia; there may, however, be dullness to percussion over the base of the lung and tenderness may be elicited immediately below the costal margin. A skiagram in the erect posture is most helpful in diagnosis since the diaphragm will be shown to be raised and a fluid level is, in most cases, demonstrable in the abscess. If in doubt, aspiration below the level of the pleural reflexion, either posteriorly or in the axillary line, may be attempted cautiously, but means to drain the abscess if pus is found should be available, and they should be put into effect at once. *Subphrenic abscess*

(5) Differential diagnosis

(a) *Spreading peritonitis*

The diagnosis of spreading peritonitis arising as a complication of an intra-abdominal inflammatory lesion does not as a rule present difficulty, but in an established case it is not always easy, or even possible, to diagnose the causative lesion. Acute pancreatitis may simulate peritonitis and an estimation of the serum amylase will be of value. As in so many cases of inflammation of an organ which has a peritoneal covering, it is impossible to state exactly when the inflammation reaches the surface and peritonitis commences, but if there is perforation of a viscus, for example, in gangrenous appendicitis, the spread of peritonitis will suddenly become more rapid and the classical signs will manifest themselves. *Acute pancreatitis*

Peritonitis following an operation will often present difficulties, for here the usual signs and symptoms are often lacking, and a rising pulse rate may be the only indication.

If the patient is treated as though peritonitis were present. If peritonitis is present, the abdomen will be more tender, vomiting more persistent and the pulse of poorer volume than in the cases of ileus alone, and the general appearance of the patient will give rise to greater anxiety.

(b) *Localized abscess*

The only other intra-abdominal cause of persistent fever following an inflammatory lesion is portal pylephlebitis which, however, is usually associated with rigors, but infection may have been carried by the blood stream to other parts of the body, and inflammation of the urinary tract, pleura or pericardium should be searched for if there is no evidence of an intraperitoneal abscess. *Portal pylephlebitis*

If an abscess fails to heal rapidly after adequate drainage the possibility of an actinomycotic infection must be considered and the discharge examined accordingly.

(6) Complications

*Paralytic
ileus*

The complications of spreading peritonitis are mainly those of the primary disease, but most cases of such peritonitis are accompanied by some degree of paralytic ileus. In the first instance this is a defence mechanism assisting in localizing the infection and, with the modern treatment of spreading peritonitis, the ileus rarely assumes serious proportions.

As localization occurs, fibrin and adhesions form between coils of small intestine, the omentum and the parietal peritoneum. These adhesions may cause organic obstruction to the small intestine, and the presence of persistent vomiting and constipation, associated with colicky pain during the resolving stage of a localized peritonitis, should lead to the suspicion of such obstruction; visible peristalsis may be observed, and increased intestinal sounds heard on auscultation. If an abscess is drained, such obstruction may clear up spontaneously and conservative treatment of intestinal obstruction has a place in such cases; careful watch is necessary, however, and if this obstruction is not relieved in a few days, laparotomy should be undertaken.

A later complication of acute peritonitis is obstruction by adhesions, months or years afterwards; this may be either subacute obstruction by massive adhesions, or acute obstruction, perhaps with strangulation by solitary adhesions which have been drawn out into bands.

(7) Treatment

(a) *Spreading peritonitis*

At this stage the treatment is that of the cause. Usually operative treatment is indicated, but in cases in which localization is the rule, for example, in peritonitis due to cholecystitis, conservative measures may be employed; such cases, however, need careful watching and if the peritonitis continues to spread, laparotomy is necessary.

(b) *Generalized peritonitis*

*Conservative
measures*

When peritonitis has become generalized, operative treatment carries a very high mortality and there is no doubt that conservative measures offer greater hope of recovery. These measures consist in:

- (i) Maintaining fluid balance by intravenous saline.
- (ii) Resting the stomach and relieving distension by indwelling stomach tube and continuous gastric suction.
- (iii) Chemotherapy. Penicillin in large doses, 50,000 units 3-hourly at first, and sulphonamides, if necessary given intramuscularly, undoubtedly have some value and should be administered in all cases.

heat cradle or hot-water bottles.

If the patient survives the acute phase, one or more localized abscesses will be formed, which will require drainage as soon as the patient is well enough. It is not always realized that such abscesses are often multiple and, if the drainage of one is followed by a continuation of the signs of toxæmia, further search must be made for another abscess.

(c) *Localized abscess*

A localized peritoneal abscess should always be drained, but provided that the patient's condition is good, and that the abscess is not increasing in size, this operation may be delayed in the case of an abscess in the lower half of the abdomen, because such abscesses sometimes spontaneously rupture into the alimentary canal and thus cure themselves. *Unless the rupture is into the rectum, the discharge of pus in the stools is not usually recognizable to the naked eye.* The method of drainage depends upon the site of the abscess; a pelvic abscess may be drained through the anterior wall of the rectum or the posterior fornix of the vagina; an iliac abscess should be drained through an incision over the point at which the abscess is nearest to the surface, but in draining such an abscess the free portion of the peritoneal cavity should be avoided—thus, if the abscess is retrocaecal, an incision in the flank is better than one in the anterior abdominal wall. Subhepatic abscesses placed anteriorly should be drained through an incision just below and parallel to the costal margin, and the same applies to the rarer anterior type of subphrenic abscess. More usually a subphrenic abscess is situated posteriorly, and here excision of the twelfth rib and drainage through the rib bed gives the most accessible and dependent drainage. In considering the time for which drainage should be continued in cases of subphrenic abscess, the same rules should apply as in drainage of an empyema, and radiography with an opaque medium in the abscess cavity should be employed to make sure that the cavity is obliterated before the drainage tube is removed.

Methods of drainage

3. TUBERCULOUS PERITONITIS

(1) *Aetiology*

This may occur at any age but is more common in children and young adults; thus, among 22 patients admitted to hospital with this condition more than half were in the second or third decades. It is always secondary to tuberculosis elsewhere but the primary focus may not be active, although miliary tubercles will be found in the peritoneum in generalized miliary tuberculosis. The infection may reach the peritoneum by the blood stream as, for example, when it comes from the lungs, but more often it occurs as the result of local spread either from mesenteric lymph glands, or from the female genital tract. The incidence is greater in females, who outnumber males by two to one.

Local spread of infection

(2) *Morbid anatomy*

Apart from generalized miliary tuberculosis there are three forms; (a) acute, (b) ascitic, and (c) plastic, but these are not strictly differentiated since the acute form may progress to either of the other two, and the plastic type may present acute symptoms due to intestinal obstruction, the ascitic and plastic types may coexist, there being loculi of fluid separated by plastic adhesions. The disease commonly involves the whole of the peritoneal cavity but may be localized to one portion, usually to the pelvis.

In the acute form the peritoneum is seen to be studded with miliary tubercles. As time goes on some of these coalesce while others resolve, and an effusion forms or the adjacent peritoneal surfaces become adherent. As a rule, the effusion is serous but occasionally it is haemorrhagic. When a

collection of tuberculous pus is found, it is always localized and associated with active disease of an adjacent organ such as a lymph gland.

(3) Clinical picture

(a) *Acute type*

This type has a sudden onset and mimics other acute abdominal inflammatory lesions, so that the abdomen may be opened under the suspicion of spreading peritonitis from other causes. Pain, vomiting and tenderness are present, together with fever, and at operation a little free fluid is found, with numerous miliary tubercles in the peritoneum. The prognosis in this type is good provided that there is not an active lesion elsewhere.

(b) *Ascitic type*

Here there may have been an acute onset but more often the condition develops very slowly with abdominal discomfort and gradually increasing distension; the patient is usually rather wasted and there are signs of free fluid. There may be some difficulty in the differential diagnosis of this type from malignant disease of the peritoneum, and in difficult cases the peritoneoscope is a valuable means of determining the nature and extent of the disease.

(c) *Plastic type*

The disease in these patients may present as a surgical emergency with acute intestinal obstruction, but more commonly it assumes a chronic form; there is abdominal discomfort often with colicky pains and, on examination of the abdomen, swellings, either obvious or ill-defined, may be felt. These swellings are due to localized collections of fluid, distended loops of intestine or thickened omentum which may be felt as a transverse tumour running across the abdomen. In extreme cases the whole peritoneal cavity may be obliterated. In this type of the disease the prognosis is more serious in view of the ever-present danger of acute intestinal obstruction, which complication is the commonest cause of a fatal termination to this disease.

(4) Diagnosis

Diagnosis is often difficult, but an active tuberculous lesion elsewhere may lead to its being suspected. In children a Mantoux test is helpful, and in the ascitic form examination with the peritoneoscope is invaluable, for miliary tubercles will be seen scattered about the peritoneum of the abdominal wall and viscera (see Plate IV).

(5) Treatment

This is mainly on general lines, and these patients benefit greatly by sanatorium treatment. In the ascitic type, the fluid should be evacuated; this can conveniently be done at the same time as a peritoneoscopy. Other surgery is only necessary to deal with obstructive complications; when these occur operation must be undertaken. If the obstruction is caused by a single band, division of the latter is all that is necessary unless the intestine is gangrenous, but more often the obstruction is associated with massive adhesions and a short-circuiting operation may be the only possibility.

Malignant
disease

Acute
intestinal
obstruction

PERITONEOSCOPY



Peritoneoscopic appearance in a case of tuberculous peritonitis, showing coalescing military tubercles on the parietal peritoneum, and a single tubercle on the small intestine.

PLATE IV

4. MALIGNANT DISEASE OF THE PERITONEUM

Malignant disease of the peritoneum is the result of extension of disease from intra-abdominal organs or from the abdominal wall. The peritoneum becomes covered with nodules of malignant growth of varying size which give rise to an effusion which may be blood-stained. It is always worth having such an effusion examined for malignant cells if the diagnosis is in doubt, but the peritoneoscope may be useful in diagnosis. In children it occurs in association with retroperitoneal sarcoma, whereas in adults it may be due to carcinoma of any intra-abdominal organ or to spread from elsewhere, for example, from the breast. The prognosis is hopeless, but these patients are much relieved by tapping, which may have to be repeated as often as every few weeks.

Blood-stained effusion

5. PSEUDOMYXOMA PERITONEI

This is a condition in which the peritoneal cavity slowly fills up with mucinous-looking fluid. It is a rare condition, but practically all recorded cases fall into one of two categories; (1) secondary to appendicitis and (2) secondary to ovarian cysts. In the former case, a mucocele of the appendix has ruptured, the base of the appendix being obstructed as a result of former inflammation; the appendix continues to secrete mucus and this escapes into the peritoneum which slowly becomes distended. In the second group a ruptured pseudo-mucinous ovarian cyst is the cause. It produces slow, painless abdominal distension, and it must be rare for the diagnosis to have been made except on laparotomy. The treatment consists in evacuation of the exudate and removal of the causative lesion.

6. TALC GRANULOMA

It is only in recent years that the importance of talc as the cause of intra-peritoneal adhesions has been fully realized. Talc, which is a silicate of magnesium, has been used for many years as a lubricant for rubber gloves; it is an insoluble powder and, like other silicates, when introduced into the tissues it sets up an inflammatory reaction with the formation of granulomas; if occurring in the peritoneal cavity, these granulomas cause adhesions which, in turn, may lead to intestinal obstruction. A careful experimental study of this problem was made by Seelig, Verda and Kidd (1943), who found that of a number of substances tried as glove lubricants, potassium bitartrate was almost as good as a dusting powder yet, being completely soluble, was absorbed, and so caused no intraperitoneal reaction.

Inflammatory reaction

Roberts (1947) has recently recorded 2 patients with talc granuloma in appendicectomy scars, and 5 patients who had thickening of the Fallopian tubes with or without adhesions, in whom talc granuloma was found. In all these cases there had been a previous appendicectomy 11-17 years before. The use of talc as a glove lubricant should therefore be abandoned and potassium bitartrate substituted.

7. ASCITES

Excess of fluid in the peritoneal cavity may be due to the following causes.

(a) Serous effusions

(i) Venous congestion in the portal circulation as a result of (a) pressure on the portal vein, for example, by malignant lymph glands, (b) cirrhosis

of the liver, and (c) back pressure in the cardiac failure of constrictive pericarditis.

(ii) Chronic nephritis.

(iii) Peritonitis, either (a) tuberculous or (b) acute or chronic inflammation of organs which have a peritoneal covering.

(iv) Malignant disease of the peritoneum.

(b) *Haemorrhagic effusions*

(i) Malignant disease of the peritoneum.

(ii) Acute pancreatitis.

(iii) Thrombosis of vessels supplying an intraperitoneal organ, for example, mesenteric thrombosis, strangulated intestine or torsion of an organ.

(c) *Chylous effusions*

These are rare. Rupture of the receptaculum chyli is usually the result of obstruction of the thoracic duct.

Treatment

When abdominal distension by ascites causes inconvenience and does not react to medical measures, tapping is necessary. With the bladder empty, the trocar is usually inserted in the midline half-way between the umbilicus and the pubes, or at either side lateral to the deep epigastric vessels. In patients with cardiac failure the effusion should only be removed slowly, but in other cases its rapid evacuation causes no ill effects.

*Portal
back pressure*

In cases of portal back pressure due to alcoholic cirrhosis, the Talma-Morrison operation, which consists in causing adhesions between the liver and the abdominal wall, and bringing the omentum out between the layers of the abdominal wall in order to create a collateral circulation, has been employed in suitable cases for many years. As a result of the work of Blakemore and Lord (1945) an alternative method, the formation of a direct anastomosis between the portal and systemic circulations, is now sometimes practised, but it is still too early to evaluate the results.

REFERENCES

- Blakemore, A. H., and Lord, J. W. (1945). *Ann. Surg.*, **121**, 435.
 Chesterman, J. T. (1946). *Brit. med. J.*, **1**, 830.
 Roberts, G. B. S. (1947). *Brit. J. Surg.*, **34**, 417.
 Seelig, M. G., Verda, D. J., and Kidd, F. H. (1943). *J. Amer. med. Ass.*, **123**, 950.
 Spalding, J. E. (1946). *Lancet*, **1**, 643.

[References to other titles are given under Peritoneum and Peritonitis in the Index Volume. The subject is also dealt with under the headings of Peritoneum: Non-inflammatory Diseases and Peritonitis in the *British Encyclopaedia of Medical Practice* (1938), Vol. 9, pp 528 and 537.]

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instances it has been possible to give an answer to many of the questions which have been asked by the public.

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